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THE DIFFERENTIAL DIAGNOSIS OF JAUNDICE WITH SPECIAL REFERENCE TO ITS SURGICAL TREATMENT*

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Fortunately, the patient with jaundice does not constitute a problem of surgical emergency except in the rare case of empyema of the gall bladder, and operation can be delayed for a sufficient length of time, perhaps 10 to 14 days, to permit a thorough attempt to establish the cause of the jaundice. Hasty, unorganized study of the problem is both unnecessary and unwise. A surgical procedure carried out on a patient with severe liver disease can be the cause of irreversible liver failure resulting in the death of the patient. On the other hand, prolonged delay in operation on a patient whose jaundice is the result of obstruction of the extrahepatic ducts may be likewise extremely harmful and may lead to more serious liver impairment or to an inoperable situation.

A careful history and a thorough physical examination are more valuable than any laboratory studies. Nevertheless, in recent years a number of liver function tests have been devised which have proved most helpful with regard to the pathologic physiology of the liver in a general sense and with regard to the differentiation between the various types of jaundice.

The amount of bile pigment, bilirubin, normally present in human serum varies between 0.2 to 0.8 mg. per 100 cc. When this bile pigment is present in excessive amounts in the blood (hyperbilirubinemia) it escapes into the tissues, *e.g.*, skin, mucous membranes, and conjunctivas, which then become stained with a yellow tint.

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Jaundice or icterus may result from (1) the production of bile pigment in excess of the amount with which the excretory power of the normal liver can cope, (2) the failure of a damaged liver to excrete the bilirubin produced in normal amounts, or (3) external obstruction of the hepatic at common bile ducts, as listed in the following outline:

Causes of Jaundice

I. Excessive production of bile pigment—normal liver
   A. *Hemolytic jaundice*—any condition which increases red cell destruction also increases the formation of bile pigment
      1. Hemolytic agents of all sorts
         a. Icterus neonatorum
         b. Congenital hemolytic anemia (acholuric or familial jaundice)
         c. Chemical poisons, *e.g.*, arsenic, ricin, etc.
         d. Toxins of certain infections, *e.g.*, septicemia
         e. Such states as malaria, pernicious anemia, and pulmonary infarction

II. Normal production of bile pigment—damaged liver
   A. *Hepatocellular, toxic* or *infective, parenchymatous*, or *retentive jaundice*
      1. Liver damage of all sorts
         a. Acute and chronic liver disease, *e.g.*, cirrhosis, infectious hepatitis, homologous serum jaundice, suppurative cholangitis, and acute yellow atrophy
         b. Various poisons, *e.g.*, arsphenamine, phosphorous, chloroform, carbon tetrachloride, etc.

c. Toxins and various pathogenic bacteria
d. Engorgement of the hepatic vessels as a result of cardiac failure

III. Obstruction of extrahepatic bile passages
A. *Obstructive* or *regurgitative jaundice*
1. Gallstones
2. Carcinoma of the head of the pancreas, Vater's ampulla, common duct, or gall bladder
3. Surgical injury of the ducts
4. Inflammatory obliteration of the ducts

It must, of course be appreciated that jaundice is frequently due to a combination of causes. For example, in obstructive jaundice the liver cells may suffer damage from prolonged retrograde pressure, and in certain cases of hepatitis the bile passages may become obstructed with mucus and other secretions. Any condition of the liver which will produce pathologic changes in one function of the liver will ultimately cause enough damage to the parenchyma of the liver to produce changes in other functions. The average length of time for this secondary damage to become manifest is from two to four weeks. Hence, liver function tests are of greatest aid in the differential diagnosis of jaundice during the initial two weeks of the disease. After this time, all the tests tend to give positive results to a varying degree, and, hence, the specificity of the various tests is lost as is their diagnostic value. For this reason, liver function tests for the differential diagnosis of jaundice should be performed as early in the course of the disease as possible.

It is obviously important to know which of these three causes is operating in any patient suffering with jaundice if proper therapy, medical or surgical, is to be applied. In such a study the evaluation of the clinical history, the physical findings, and the laboratory tests must be closely correlated with a thorough understanding of the normal physiology of the liver with its multiplicity of functions and its behavior in pathologic states.

As a matter of practical importance the diagnosis boils down to the differentiation between some form of hepatitis (including cirrhosis) on the one hand, *i.e.*, hepatocellular or parenchymatous jaundice, and extrahepatic biliary obstruction on the other: differentiation, therefore, between groups II and III of our classification. Group I (hemolytic jaundice) is usually not a difficult diagnostic problem in the deeply icteric patient. In these cases there is usually a familial history, an enlarged spleen, and red cell fragility.

TABLE 1

Final diagnoses formed in 146 consecutive cases of jaundice

Diagnosis	No. of Cases
Cirrhosis of the liver	49
Infectious hepatitis	43
Carcinoma of the pancreas	11
Cholelithiasis	9
Acute cholecystitis	6
Undiagnosed	5
Stricture of the common duct	4
Lobar pneumonia	4
Chronic cholecystitis	3
Acute pancreatitis	2
Lymphosarcoma of the liver	2
Metastatic carcinoma of the liver	1
Primary carcinoma of the liver	1
Abscess of the liver	1
Pancreatic cysts	1
Acute cholangitis	1
Hodgkin's disease	1
Carcinoma of the gall bladder	1
Carcinoma of the ampulla	1
Total	146

Final diagnoses in 146 consecutive cases of jaundice in the Hospital for the Women of Maryland, previously reported by us,[5] excluding the jaundice occurring in infancy, are summarized in table 1. Sixty-three per cent of these jaundiced women had liver damage as a primary cause, whereas the great majority of the remainder were in the extrahepatic obstruction group.

HISTORY

In almost no other condition can the medical history of an individual be more important in arriving at the cause than in jaundice. A familial history of hemolytic anemia with or without gallstones is very important in the elimination of hemolytic (prehepatic) jaundice. One should find out immediately if the patient has traveled in certain areas in which specific diseases are endemic; if there has been any blood transfusion, vaccine, or serum given in the recent past; if there has been any recent exposure to patients suffering with jaundice; if the patient's occupation leads to contact with hepatotoxic fumes or

gases; if there is a history of ingestion of any of the numerous drugs which lead to liver damage such as mercury, arsenic, cinchophen, carbon tetrachloride and sulfonamide groups; if there is a history of excessive alcohol intake; and if there has been any previously existing disease of the stomach, colon, pancreas, gall bladder, spleen, or liver which might now be resulting in widespread metastases.

In connection with the homologous serum jaundice referred to as a possibility in the last paragraph, this complication will follow blood transfusion in about 4 per cent of the patients and will follow the parenteral use of pooled plasma in a much higher incidence. In fact, during the recent Korean conflict 25 per cent of all those receiving plasma developed homologous serum jaundice. This whole question is a fascinating problem in itself and a solution is now being sought in various ways, including the elimination of the use of a large pool of donors, the chances of encountering blood infected with the jaundice virus being, of course, less in inverse proportion to the number of donors to the pool. A splendid contribution to this subject has been made by Allen and his associates,[1] who have prevented the occurrence of homologous serum jaundice following the administration of pooled blood plasma by storing the plasma at 78 to 96° F. for six months before giving it to the patients. This work should have great practical significance in military operations or in large scale civilian disasters.

A history of the possible accidental inhalation of carbon tetrachloride is very important because this substance is used so widely in cleaning fluids. Recently 2 cases of severe central necrosis of the liver with jaundice due to this cause have been called to my attention.[2]

SYMPTOMS

1. Pain. Painless progressive jaundice in middle aged patients with acholic stools is usually due to a malignant lesion blocking the extrahepatic ducts, unless the patient has had a previous operation on the biliary tract, in which case it may be due to a stricture of the common or hepatic ducts. Therefore, in general, we have come to regard pain in jaundice as being associated with a stone and an absence of pain as being associated with cancer, usually at the head of the pancreas. How often is this true?

Malignancy was responsible for one-third of 100 consecutive jaundiced patients operated on in a series of cases reported by Zollinger and Saleeby.[10] However, only 1 out of 3 patients with malignancy in this group had what might be termed painless jaundice. More than one-half had some bizarre upper abdominal pain which sometimes was quite severe. Therefore, more diagnostic significance should be attached to persistent jaundice of increasing intensity rather than to the presence of pain.

In a recent study of 175 patients who were subjected to operation and in whom common duct stones were found,[5] only 79 per cent had biliary colic; 21 per cent had no pain. In another series of 100 jaundiced patients subjected to surgery,[9] 70 per cent of those with common duct stones gave a typical history of biliary colic, whereas 30 per cent had none. Since common duct stones are responsible for approximately one-half of all cases of obstructive jaundice requiring operation, this variation in the occurrence of pain is important to recall.

2. Mode of onset and persistence of jaundice. Persistent jaundice of increasing intensity is indicative of a malignant lesion and is of greater diagnostic significance than the absence of pain. The development of jaundice immediately following cholecystectomy strongly suggests injury of the common bile duct, an overlooked stone, or a late reaction to a blood transfusion. Pancreatitis should be ruled out by an amylase determination. In the late postoperative period following cholecystectomy, an overlooked stone is the most likely cause of jaundice.

Mention should be made at this point that jaundice is not to be relied on in establishing the diagnosis of common duct stone. Too frequently the surgeon does not explore the common duct for calculi unless the patient is jaundiced at the time of operation. In a series of 100 consecutive cases in which a common duct stone was recovered at operation,[9] there was no history of jaundice in 35 per cent, a past history in 21 per cent, and only 44 per cent were jaundiced at the time of operation.

3. Nausea and vomiting. Nausea and vomiting are common in the presence of common duct stones and uncommon with obstruction of the common duct due to a tumor.

4. Shock. Attention should be called to the extreme prostration, sometimes amounting to the most severe shock, which can occur in elderly patients when a stone blocks the common duct

completely and suddenly. Jaundice may or may not be present, depending on the ball valve action of the stone. The fever is very high and the pulse is rapid. These patients should be regarded as surgical emergencies. Multiple tiny abscesses in the liver as a result of bile blockage by the stone have been found in some of these cases.

5. Chills and fever. Such a symptom complex, sometimes called "Charcot's fever," often accompanies stone or stricture which partially block the common duct and lead to an ascending cholangitis. Regurgitation of gastric juice into the hepatic ducts, as when the stomach or duodenum are anastomosed to the gall bladder or to the common duct, may cause the same symptoms. These symptoms are uncommon in hepatocellular jaundice or jaundice due to tumor blocking the common duct.

PHYSICAL EXAMINATION

Among the important findings are: weight loss; the intensity of the jaundice, which is apt to be greater in malignancy; the presence or absence of small hemangiomas in the skin; the presence or absence of gynecomastia; and the presence or absence of an enlarged liver, spleen, or gall bladder.

Estrogens are esterified in the liver, and in liver disease this esterification may be so impaired that gynecomastia may result from the presence of excess estrogen.

Since the liver is usually enlarged and tender in the presence of jaundice of most types, its size contributes little to the differential diagnosis unless distinct metastatic nodules can be palpated. On the other hand, an enlarged spleen increases the diagnostic possibilities considerably and serves as a danger signal to the surgeon. Congenital and acquired hemolytic anemia and cirrhosis of the liver must be considered in these cases and extensive hematologic investigations carried out.

The finding of an enlarged gall bladder, especially in the presence of jaundice, is of considerable importance and is a point commonly emphasized and referred to as Courvoisier's law. This has been so variously quoted and misquoted that I went to the library to seek enlightenment. To my surprise I learned that Courvoisier was not a Frenchman, but was of German-Swiss extraction. His full name was Ludwig G. Courvoisier. He lived from 1843 to 1918 and was professor of surgery at the University of Basel, Switzerland. His statement, which has become known as Courvoisier's law, appeared in 1890 in his book on the pathology and surgery of the gall bladder, published in Leipsig.[3] See figure 1.

A literal translation of Courvoisier's law into English states: "In stone obstruction of the common bile duct, dilation of the gall bladder is rare; the organ is previously already usually contracted. In obstruction of another kind dilation is on the contrary the customary finding (atrophy occurs in only $\frac{1}{12}$ of these cases)."

In our experience a distended gall bladder is palpable in about two-thirds of the patients with carcinoma of the head of the pancreas or of Vater's ampulla. The absence of a distended gall bladder in the presence of jaundice favors the diagnosis of a common duct stone because of the contraction due to repeated infections, as pointed out by Courvoisier. Lastly, a palpable gall bladder in the absence of jaundice is indicative of a hydrops from a stone occluding the cystic duct.

LABORATORY EXAMINATION

The first two tests to be made in the laboratory on patients with jaundice are those on stool and urine. Both are dependent on abnormalities of bile secretion or elimination. One of the main functions of the liver is the secretion of bile, the chief components being bile salts, bile pigments, cholesterol, and lecithin.

Stool. Probably the most important and yet the most neglected examination of a patient with jaundice is the simple determination of whether or not there is bile pigment in the stools. Many times the most complicated liver function and other tests are recorded on the patient's chart,

Bei Steinobstruction des Choledochus ist Ectasie der Gallenblase selten; das Organ ist vorher schon gewöhnlich geschrumpft. Bei Obstruction andrer Art ist dagegen Ectasie das Gewöhnliche; Atrophie besteht nur in $^1/_{12}$ dieser Fälle.

FIG. 1.

but no record is made of whether or not the stools were bile colored; yet this fact could be established by a digital rectal examination alone, and the presence of an obstructive or a nonobstructive jaundice could thus be determined at once.

Clay colored stools imply an absence of bile due to complete obstruction of its flow; the finding of stools consistently so discolored calls strongly for surgical intervention.

Urinary urobilinogen. In the first series of jaundiced patients reported by us,[5] it was observed that one of the most helpful and accurate laboratory tests was the determination of urinary urobilinogen. It will be recalled that when bile reaches the intestine, it is transformed into urobilinogen. Some is excreted in the stool and the rest is reabsorbed and carried to the liver in the portal blood stream. Some of this is re-excreted in the bile and some is absorbed in the general circulation and eliminated through the kidneys. Thus, in obstructive jaundice very little bile reaches the intestine, hence little or no urobilinogen is formed and accordingly little or none is found in the urine (normal, 0 to 4 mg. per day). However, in hepatocellular jaundice with diffuse hepatic disease, it is assumed that adequate bile reaches the intestinal tract for conversion into urobilinogen which is absorbed into the portal system, but the re-excretion by the liver is impaired. Accordingly, a much greater amount of urobilinogen is then conveyed to the kidneys for excretion. Perhaps the most marked derangement of urobilinogen metabolism is present in hemolytic disorders, which cause great amounts of bilirubin to be excreted by the liver and, hence, much more urobilinogen in the urine and the stools (fig. 2).

The following four tests are useful to indicate the intensity of the jaundice and the degree of liver damage, but, except for the thymol turbidity test, are not helpful in the differential diagnosis of jaundice: (1) serum bilirubin, (2) Bromsul-

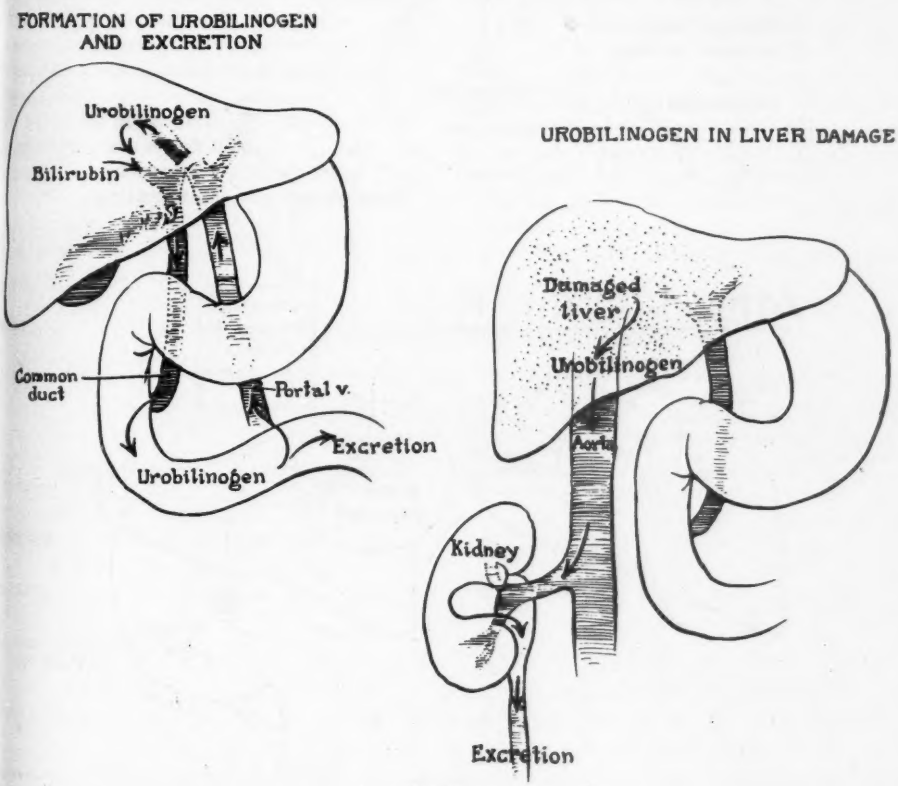

FIG. 2.

phalein, (3) serum albumin and globulin, and (4) thymol turbidity.

The minimal program of tests for differentiation of obstructive and parenchymal jaundice at the Johns Hopkins Hospital are (1) urinary urobilinogen, (2) plasma prothrombin time, (3) prothrombin tolerance, (4) cephalin cholesterol flocculation, (5) thymol turbidity, and (6) serum alkaline phosphatase.

Liver function tests. It is necessary to resynthesize one's knowledge of liver physiology before one can properly understand and evaluate liver function tests. The liver, of course, has a multiplicity of functions and is important in the metabolism of proteins, carbohydrates and lipids, in the formation and excretion of bile, and in the conjugation and destruction of numerous natural and synthetic substances, including alkaline phosphates and estrogens.

In the metabolism of proteins within the liver, fibrinogen, prothrombin, and albumin are synthesized. Clinically, a fall in plasma fibrinogen is rare except in the presence of catastrophic liver disease such as acute yellow atrophy. The fall in fibrinogen manifests itself in prolongation of the clotting time, and in the tendency to bleed. A fall

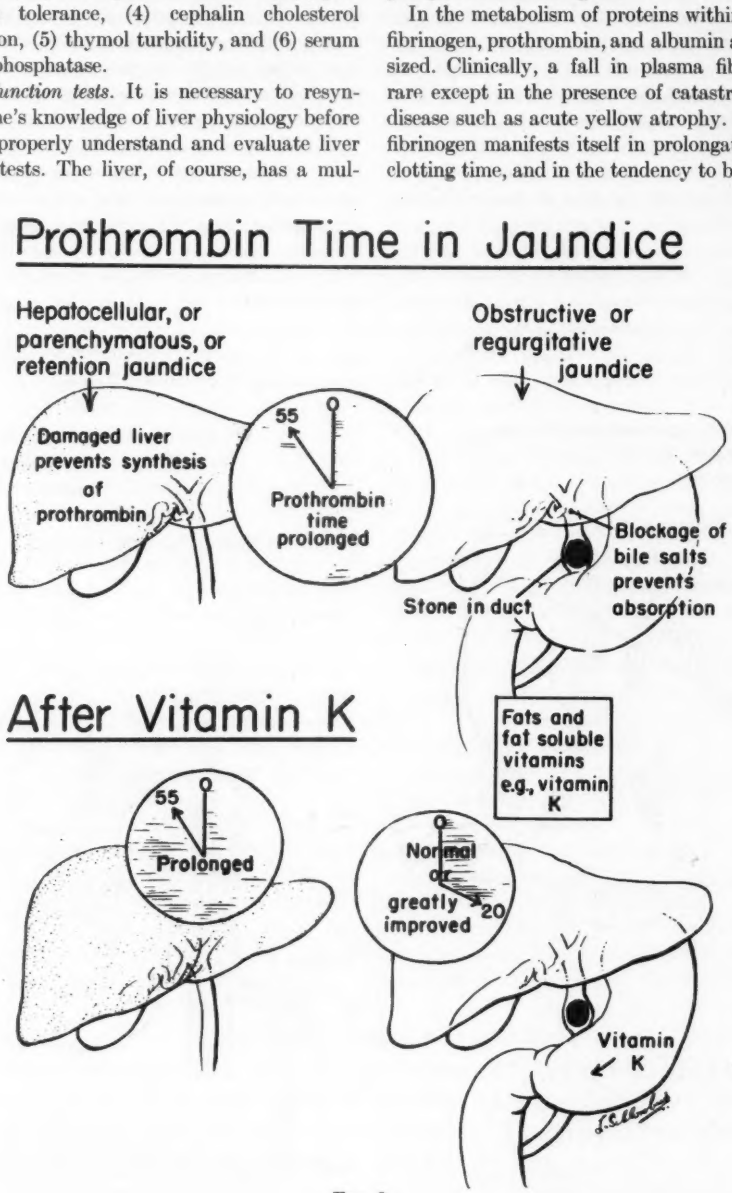

FIG. 3.

in the serum albumin level, on the other hand, is common in hepatic disease and results in a decline in the plasma colloid osmotic pressure, which may in time dispose to the accumulation of ascitic fluid and edema. A hemorrhagic tendency in liver disease may at times be attributed to the decrease in plasma prothrombin.

Plasma prothrombin time and prothrombin tolerance. It is very important in this connection to understand that the prolongation of prothrombin time in this case is apparently due to a failure of synthesis of prothrombin by the liver and must be differentiated from the prolongation of prothrombin which occurs in obstructive jaundice as the result of failure of absorption of vitamin K from the small intestine. Such differentiation is involved in one of our most dependable laboratory tests on jaundiced patients and can be made by intravenous or intramuscular injection of 1 or 2 mg. of synthetic vitamin K.

In the patient with obstructive jaundice the prolonged prothrombin time will be returned to normal almost immediately. When more than a moderate amount of liver damage is present, however, the administration of vitamin K parenterally is followed by little or no shortening of the prolonged prothrombin time. Rarely should a patient be accepted for surgery whose prothrombin time will not become elevated well above 50 per cent in response to the administration of vitamin K.

If vitamin K is administered to patients in whom the prothrombin time is 50 per cent of normal or less, by giving 2.4 mg. of 2-methyl-1,4-naphthoquinone-3-sodium sulfonate (Hykinone) intramuscularly, and repeating the test for prothrombin time, this time will be shortened and will usually rise rapidly to normal in the patient with obstructive jaundice. Where there is more than a moderate amount of liver damage, however, the administration of vitamin K is followed by little or no shortening of the prolonged prothrombin time (fig. 3).

Cephalin flocculation and thymol turbidity tests. In a recent study at Johns Hopkins Hospital,[4] 127 jaundiced patients were studied to clarify the interpretation of the three liver function tests which were found to be the most helpful in the differentiation between parenchymal liver disease and extrahepatic biliary obstruction. In each case the cause of the jaundice was clearly established by operation, autopsy, or prolonged observation. These tests are cephalin flocculation, thymol turbidity and alkaline phosphatase.

The cephalin flocculation and the thymol turbidity tests are most commonly used to reveal disease of the hepatic cells because they are relatively simple and have a broad reference of comparison in many hospitals. Both of these tests are based not only on the fact that the serum globulin levels are increased in the course of liver disease but that the qualitative composition of the globulin is changed.

In 79 of the 127 patients studied there was

TABLE 2

LIVER FUNCTION IN PATIENTS WITH PARENCHYMAL LIVER DAMAGE

DISEASE	NO. OF PATIENTS	NO. WITH CEPHALIN FLOCCULATION GREATER THAN 4+	NO. WITH THYMOL TURBIDITY GREATER 5.0 UNITS PER cc.	NO. WITH ALKALINE PHOSPHATASE OF 5-10 BODANSKY UNITS	NO. WITH ALKALINE PHOSPHATASE OF 10-15 BODANSKY UNITS	NO. WITH ALKALINE PHOSPHATASE GREATER THAN 15 BODANSKY UNITS
CIRRHOSIS	35	25	28	22	7	6
HEPATITIS	34	30	32	17	9	8
SYSTEMIC INFECTIONS OTHER THAN WEIL'S DISEASE	10	9	9	9	1	0
TOTALS	79	64	69	48	17	14

parenchymal liver damage. The results of the liver function tests were as shown in table 2. The results of the remaining 48 patients, who had obstruction of the extrahepatic biliary ducts, are shown in table 3.

It is evident that in most of the patients with parenchymal liver disease the cephalin flocculation and thymol turbidity are elevated, whereas the alkaline phosphatase is normally, but not always, below 15 Bodansky units. In obstructive

TABLE 3

LIVER FUNCTION IN PATIENTS WITH MECHANICAL OBSTRUCTION.

DISEASE	NO. OF PATIENTS	NO. WITH CEPHALIN FLOCCULATION GREATER THAN +	NO. WITH THYMOL TURBIDITY GREATER THAN 5.0 UNITS PER cc.	NO. WITH ALKALINE PHOSPHATASE OF 5-10 BODANSKY UNITS	NO. WITH ALKALINE PHOSPHATASE OF 10-15 BODANSKY UNITS	NO. WITH ALKALINE PHOSPHATASE GREATER THAN 15 BODANSKY UNITS
CARCINOMA - -	25	6	10	1	6	18
GALLSTONE - - -	20	6	7	2	5	13
STRICTURE - - - -	3	1	1	0	0	3
TOTALS - - - - -	48	13	18	3	11	34

TABLE 4

COMPARISON OF LIVER FUNCTION TESTS IN OBSTRUCTIVE AND NONOBSTRUCTIVE JAUNDICE.

CAUSE OF JAUNDICE	NO. OF PATIENTS WITH CEPHALIN-FLOCCULATION OR THYMOL-TURBIDITY DETERMINATIONS (OR BOTH)		NO. WITH ALKALINE PHOSPHATASE BELOW 15 BODANSKY UNITS	NO. WITH ALKALINE PHOSPHATASE ABOVE 15 BODANSKY UNITS
	ELEVATED	NORMAL		
PARENCHYMAL LIVER DISEASE	69	10	65	14
MECHANICAL OBSTRUCTION	18	30	14	34

jaundice the majority of the cephalin flocculation and thymol turbidity values are normal, and in most cases the alkaline phosphatase is above 15 (table 4).

Serum alkaline phosphatase. Alkaline phosphatase, produced in bone and liver, is excreted in the bile. Credit for early recognition of the association between an elevated blood phosphatase and jaundice, and the diagnostic significance thereof, must go to Robison[7] (1923) and Roberts[6] (1933). The serum concentration of alkaline phosphatase was between 1.5 and 5.4 Bodansky units in 200 consecutive patients studied at the Johns Hopkins Hospital without evidence of disease affecting bone or liver. Elevations above this normal may result from several factors. Obstruction of the biliary ducts will elevate the serum alkaline phosphatase level by interfering with its excretion. This rise may occur when there is only partial obstruction of the biliary outflow.

When obstruction of the biliary ducts is associated with significant liver cell damage the elevation of the serum alkaline phosphatase may be less marked than it is in pure obstruction without parenchymal damage, because there is decreased production of alkaline phosphatase by the liver. This is a very important consideration and should serve as a warning to the alert surgeon that such a patient may be too ill for operation at this stage, as pointed out by Mellinkoff and co-workers.[4] Another condition in which an elevation of serum alkaline phosphatase above normal is sometimes found is in diffuse intrahepatic ducts.

In none of the cases studied was there proved common duct obstruction without severe concomitant liver disease in which, if serial determinations were made, the phosphatase remained below 15 units while the serum bilirubin was rising. It thus appears that a serum alkaline phosphatase of 15 Bodansky units is the best approximate dividing line between uncomplicated obstructive and nonobstructive jaundice. It is impossible, however, to be dogmatic about any one figure. No laboratory procedure is that reliable.

We may conclude that the serum alkaline phosphatase will frequently point to the correct cause of jaundice if the following qualifications are kept in mind: A high phosphatase unrelated to liver disease may be present (1) when there is a coincident disease of the bones, (2) in hyperthyroidism, (3) in hyperparathyroidism, and (4) in pregnancy after the fifth or sixth month. In an important minority of patients with parenchymal liver disease, notably cirrhosis or some forms of hepatitis, the alkaline phosphatase may be very high. In the presence of obstruction of the common bile duct the phosphatase may be relatively low (below 15 units) under the following circumstances: (1) when the obstruction is partial or temporary, as judged by failure of serial bilirubin determinations to show progressive elevation; (2) when coexistent parenchymal liver disease is severe; (3) very early in the course of an obstructive process; (4) and in a rare case for reasons that are not clear. Thus, *when there is progressive hyperbilirubinemia due to ductal obstruction and without serious hepatic damage, serial determinations of the alkaline phosphatase will almost invariably reveal levels above 15 units.*

The correct interpretation of the alkaline phosphatase is probably the most important laboratory aid in distinguishing parenchymal liver disease and extrahepatic obstruction. Often the cephalin flocculation and thymol turbidity tests will give added useful information. There may be some confusion when parenchymal and obstructive liver disease both are present and in some cases the laboratory findings and clinical features seem contradictory. In either case it is certainly poor judgment to base therapeutic management on the laboratory tests alone. Continued observation of the patient's progress and serial alterations in these tests will nearly always clarify the diagnosis. In an occasional case the diagnosis will still be obscure. One must then weigh the comparative hazards of continued observation, liver biopsy, and surgical exploration.

SUMMARY

1. A presentation is made of the classification of the different types of jaundice.

2. Certain clinical and laboratory methods in the differential diagnosis between those who should be treated medically and those who should be treated surgically are discussed.

3. Liver function tests to be reliable must be made very early in the disease.

4. Acholic stools, decreased in absent urobilingen in the urine, good prothrombin response to vitamin K, and elevated serum alkaline phosphatase (15 Bodansky units or more) support the diagnosis of jaundice which should be treated surgically.

5. Elevated cephalin flocculation and thymol

turbidity, and poor prothrombin response to vitamin K, support the diagnosis of jaundice which should be treated medically.

6. The correct interpretation of the alkaline phosphatase test is probably the most important laboratory aid in distinguishing parenchymal liver disease and extrahepatic obstruction.

7. In an occasional case of jaundice the diagnosis even after extensive study will remain obscure. One must then weigh the comparative hazards of continued observation, liver biopsy, and surgical exploration.

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REFERENCES

1. Allen, J. G., Inouye, H. S., and Sykes, C.: Homologous serum jaundice and pooled plasma—attenuating effect of room temperature storage on its virus agent. Ann. Surg., *138:* 476, 1953.
2. Classen, J. N.: Personal communication.
3. Courvoisier, L. G.: Casuisitisch-statistische Beiträge zur Pathologie und Chirurgie der Gallenwege. Vogel, Basle, 1890.
4. Mellinkoff, S. M., Tumulty, P. A., and Harvey, A. M.: Differentiation of parenchymal liver disease and mechanical biliary obstruction. New England J. Med., *246:* 729, 1952.
5. Pearce, W. F., and Trimble, I. R.: Jaundice; 15-year incidence report at Hospital for Women of Maryland. Maryland M. J., *4:* 355, 1955.
6. Roberts, W. M.: Blood phosphatase and van den Bergh reaction in differentiation of several types of jaundice. Brit. M. J., *1:* 734, 1933.
7. Robison, R.: The possible significance of hexosephosphoric esters in ossification. Biochem. J., *17:* 286, 1923.
8. Waugh, J. M., Johnston, E. V., and Cain, J. C.: Surgical aspects of choledocholithiasis. J. A. M. A., *154:* 734, 1954.
9. Zollinger, R. M., and Britt, C. I.: Indications for surgery in jaundiced patients. J. Kentucky M. A., *52:* 91, 1954.
10. Zollinger, R. M., and Saleeby, R. G.: Indications for surgery in jaundiced patients. J. Indiana M. A., *46:* 485, 1953.

CARCINOMAS OF THE APPENDIX

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Primary malignant tumors of the appendix are encountered infrequently, with the exception of carcinoid. The incidence is difficult to determine because of the inaccurate early literature which failed to distinguish between adenocarcinoma and tumors that we designate as carcinoid. Even as late as 1942, it was suggested that carcinoid be classified as grade 1 adenocarcinoma. Failure to distinguish between cases of adenocarcinomas which arise in the cecum and secondarily invade the appendix adds to the statistical confusion.

Uihlein and McDonald[4] reviewed the experiences in the Mayo Clinic from 1910 to 1941 and found 144 malignant lesions of the appendix. From this group 127 cases (88.2 per cent) were carcinoids, 12 (8.3 per cent) were papillary cystoadenocarcinomas (malignant mucoceles), and 5 (3.5 per cent) were adenocarcinomas of the colonic type. The same authors state that there is still considerable confusion with regards to epithelial neoplasms of the appendix, and they suggested the following classifications: (1) carcinomas of carcinoid type (malignant carcinoids), (2) carcinomas of cystic type (papillary cystoadenocarcinomas), and (3) carcinomas of colonic type (adenocarcinomas).

In reviewing 441 cases of carcinoma in the gastrointestinal tract at Crawford W. Long Hospital, Atlanta, Georgia, 171 originated in the colon, 169 in the rectum, 75 in the stomach, 9 in the small bowel, 8 in the esophagus, and 7 in the appendix. This represents an incidence of 1.5 per cent of the total gastrointestinal carcinomas treated in this hospital. At the time of this study, a review of pathologic reports of appendectomies in the last 10 years revealed 7 cases of carcinoma of the appendix. This does not include the cases of carcinoid that did not show invasion. It is generally accepted that there is a ratio of adenocarcinoma of the appendix to carcinoids of the appendix of approximately 1:10, which generally agrees with this series.

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CASE REPORTS

Case 1. Mrs. E. W. (S47-1508), an 18-year-old white woman, was admitted with a history of acute diffuse abdominal pain of 1 week's duration followed by vomiting and diarrhea 2 days prior to admission. The admitting diagnosis was acute recurrent appendicitis. A routine exploratory laparotomy was performed with appendectomy. The pathologic report was malignant argentaffinoma. The tumor was classified as malignant because it was invading the muscle layer without penetrating this layer at any point. The patient is doing well in the 10th postoperative year without any signs or symptoms of recurrence.

Case 2. Mrs. J. N. H. (S47-1471), an 18-year-old white woman, was admitted with the chief complaint of pain in the right lower quadrant, which became more severe at the time of menstruation. Associated with this complaint was irregular menstruation and occasional intermenstrual bleeding. On examination, a palpable mass was demonstrated in the right lower quadrant, which measured 4 by 4 by 4 cm. This was thought to be an ovarian cyst. At exploratory laparotomy a follicular cyst of the ovary was found and a stricture at the tip of the appendix was also noted. The tip of the appendix was enlarged, yellow in color, and firm on palpation. An appendectomy was performed and the pathologic report was "malignant carcinoid" of the appendix. This patient is now living and well 11 years following surgery.

Case 3. Mrs. O. N. A. (S57-6637), a 68-year-old white woman, was admitted to the hospital with a history of pain of 4 days' duration, in the right lower quadrant associated with nausea and vomiting. The abdomen was tender in the right lower quadrant, with muscle spasm and no peristalsis. No mass or tumor was demonstrated. Leukocytes were 18,800 with 81 per cent polymorphonuclear leukocytes. A preoperative diagnosis of acute appendicitis was made and the patient was explored on the day of admission. An acute perforated appendix was found and a routine appendectomy was performed. The pathologic report was adenocarcinoma of the appendix, low grade, without evidence of invasion, and acute suppurative appendicitis with periappendicitis and perforation. The pathologist's report further stated that the perforation

was probably distal to the mucosal neoplasm. This patient was reexplored 3 months after her initial surgery and no tumor was identified. She is now in good health 7 months after her initial procedure.

Case 4. Mr. A. M. C. (S49-3272), a 68-year-old white man, was admitted with a history of abdominal pain, nausea, and vomiting of approximately 8 hours' duration. Leukocytes were 17,700 with 90 per cent polymorphonuclear leukocytes. The abdomen was pendulous without rigidity. There was suggestive rebound and tenderness in the right lower quadrant. Peristalsis was absent. A diagnosis of acute appendicitis was made prior to surgery. The patient was explored and a routine appendectomy was performed. The subsequent pathologic diagnosis was mucinous adenocarcinoma of the appendix. The patient, with a hemoglobin of 7.5 gm. and in a general state of emaciation, was considered a poor surgical risk. Whole blood was administered in the immediate preoperative period. Surgical exploration was limited entirely to the area of the appendix because of the patient's poor condition. He had a progressive downhill course and died on the 3rd postoperative day. Autopsy was not performed.

Case 5. Mrs. E. L. (S56-5714), a 74-year-old white woman, was admitted to the hospital with the diagnosis of right ureteral stone. On admission an intravenous pyelogram was performed and no stones were visualized. Leukocytes were 22,400. She had pain in the right lower quadrant with abdominal distention and spasm. The following day leukocytes were 20,800 with 93 per cent polymorphonuclear leukocytes. A diagnosis of acute appendicitis was made and verified at exploration. The appendix was found to be acutely inflamed, perforated, and retrocecal. No tumor in the appendix or in the cecum was described. The pathologic report was adenocarcinoma of the appendix with invasion of the appendiceal wall and perforation with extension to the proximal amputated end of the appendix.

She was discharged from the hospital on her 10th postoperative day. Six months later she developed indigestion, abdominal distention, and abdominal pain. She refused surgery and died, 1 year after the appendectomy, with generalized carcinomatosis.

Case 6. Mr. S. E. (S49-1594), a 48-year-old white man, was admitted to the hospital on April 10, 1949. In November 1948 he had had intermittent pain in the right upper and right lower quadrants of the abdomen, with intolerance to fatty foods. The abdomen was soft, and no mass was palpated; however, there was rebound tenderness in the right lower quadrant. Leukocytes were 7350 with 72 per cent polymorphonuclear leukocytes. Exploratory laparotomy was performed 9 days after admission. Exploration revealed 4000 cc. of sanguineous effusion in the abdominal cavity and a mass involv-

TABLE 1

Clinical analysis of 7 cases of carcinoma of the appendix

Case	Age	Presenting Symptoms	Procedure	Operative Findings	Pathology	Follow-up
	years					
1	18	Acute appendicitis	Appendectomy	Acute appendicitis	Malignant argentaffinoma	Well after 10 years
2	18	Right ovarian cyst or tumor	Right ooforectomy appendectomy	Appendix grossly normal	Malignant argentaffinoma	Well after 10 years
3	68	Acute appendicitis	Appendectomy	Perforated appendix	Adenocarcinoma of appendix, low grade	Well after 6 months
4	68	Acute appendicitis	Appendectomy	Perforated appendix	Adenocarcinoma of appendix, mucinous type	Died after 3 days
5	74	Acute appendicitis	Appendectomy	Perforated appendix	Adenocarcinoma of appendix	Died after 1 year
6	48	Chronic cholecystitis	Partial appendectomy	Mass involving cecum, appendix, ileum, and omentum	Malignant argentaffinoma with metastasis	Died after 72 days
7	68	Intestinal obstruction	Intestinal resection	Tumor in ileocecal area	Malignant argentaffinoma of appendix	Well after 9 years

ing the cecum, appendix, terminal ileum, and omentum. The appendix was removed with the exception of the distal end, which could not be removed because of technical difficulties. The pathologic report was malignant argentaffinoma of the appendix with metastases. Soon after the operation, the patient developed gangrene of his foot, associated with an embolus to the left popliteal artery. He was treated with sympathetic blocks and anticoagulants; response to therapy was poor. On his 63rd postoperative day he developed a fecal fistula through the upper half of the operative incision, and, following a progressively downhill course, he died on the 73rd postoperative day. At autopsy he was found to have generalized carcinomatosis with intestinal obstruction and fecal fistula. There were also metastatic lesions in the lungs.

Case 7. Mrs. E. S. L. (S49-27), a 68-year-old white woman, had been admitted to the hospital on three previous occasions and had had four laparotomies for intestinal obstruction, ventral hernia, and lysis of abdominal adhesions. On her last hospital admission a diagnosis of intestinal obstruction was made, and at exploratory laparotomy a mass in the ileocecal area was found, with necrosis of the terminal ileum. Approximately 6 inches of the ascending colon, cecum, appendix, and 100 cm. of the terminal ileum were resected at surgery. The pathologic report was malignant argentaffinoma and foreign body reaction. The patient had an uneventful postoperative course and is living and well with the exception of an incisional postoperative hernia.

DISCUSSION

The preoperative diagnosis of carcinoma of the appendix is rarely made. Generally, such patients may be grouped into five diagnostic classes: (1) The majority of these lesions are discovered incidentally at the time of abdominal surgery for other pathology. (2) Malignant lesions of the appendix are not infrequently discovered at operation performed for symptoms related to acute appendicitis. (3) Carcinoma of the appendix is discovered on physical examination as a mass in the right lower quadrant or right flank. (4) Metastatic carcinoma is identified with an adenocarcinoma of the appendix. (5) Malignant argentaffinomas are removed in an apparently grossly normal appendix and are not suspected by the surgeon at the time.

It is readily recognized that this series of patients is too small for an exhaustive analysis or for any definite conclusions (table 1). Of the 7 patients, 5 were women and 2 were men, their ages ranging from 18 to 74 years. The correct preoperative diagnosis was never made or even suspected in any patient in this group. Of the 7 cases, 4 presented the clinical picture of acute appendicitis, 1 case was diagnosed preoperatively as intestinal obstruction, 1 case was thought to have chronic cholecystitis, and the remaining case was found incidental to removal of a right ovarian cyst.

Malignant argentaffinomas can be surgically treated by a simple appendectomy if they are localized to the appendix. If there is invasion of the cecum by the argentaffinoma, then a right hemicolectomy is indicated. It is our impression that adenocarcinomas of the appendix should be treated radically and in a manner similar to carcinoma of the cecum, *e.g.*, lymph node dissection and end to end ileotransverse colostomy.

SUMMARY

The problem of primary malignant tumors of the appendix is reviewed. Seven patient reports are presented.

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REFERENCES

1. Hilsabeck, J. R., Judd, E. S., Jr., and Woolner, L. B.: Carcinoma of the vermiform appendix. S. Clin. North America, *31:* 995, 1951.
2. Lesnick, G., and Miller, D.: Adenocarcinoma of the appendix. Cancer, *2:* 18, 1949.
3. Ryan, R. F., Beahrs, O. H., Woolner, L. B., and Bartholmew, L. G.: Malignant mucocele of the appendix. S. Clin. North America, *37:* 1029, 1957.
4. Uihlein, A., and McDonald, J. R.: Primary carcinoma of the appendix resembling carcinoma of the colon. Surg. Gynec. & Obst., *37:* 711, 1943.

TRAUMATIC RUPTURE OF THE SPLEEN ASSOCIATED WITH PORTAL CIRRHOSIS, PORTAL HYPERTENSION, AND CONGESTIVE SPLENOMEGALY

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Since Celsus first reported his experience with a case of traumatic rupture of the spleen in the 15th century,[2] a significant number of reports dealing with splenic rupture have appeared in the surgical literature. Experiences with splenectomy for this condition have been documented.[4, 6, 7]

Rupture may occur in the normal and diseased spleen and may be spontaneous, traumatic, or delayed. A critical survey of the literature has revealed no documented cases of traumatic rupture of the spleen with congestive splenomegaly associated with portal cirrhosis and portal hypertension.

The purpose of this paper is to describe in detail the diagnosis and management of a case of delayed rupture of a congested spleen incident to portal cirrhosis and portal hypertension following a friendly blow of the hand to the upper abdomen. The severity of cirrhosis and the relatively normal liver function studies are of particular interest.

CASE REPORT

A 40-year-old white man was admitted to the medical service at the Jefferson Medical College Hospital on October 17, 1957, because of generalized abdominal pain, especially in the left upper quadrant of the abdomen, and left shoulder pain of 5 days' duration. Ten days previously he had developed symptoms of influenza. On October 12, 1957, he received a friendly tap of the hand over the epigastrium and left upper quadrant of the abdomen and on the following day, shortly after the evening meal, he experienced diffuse, sharp, constant abdominal pain that became localized to the left hypochondrium. There was associated pain in the left shoulder. Deep inspiration and motion accentuated the pain, but bed rest and narcotic injections by his family physician afforded partial relief. Symptoms progressed up to the time of hospital admission.

The past medical history revealed that this patient was a heavy consumer of alcohol and a diabetic, requiring regular insulin daily. The patient's father had died of massive bleeding incident to portal cirrhosis and esophageal varices. His mother had diabetes mellitus.

On physical examination his temperature was 101° F., pulse 100, respiration 24, and blood pressure 150/80 mm. of Hg. The patient was obese and appeared chronically ill. The skin was warm and the sclera was icteric. The heart rate and rhythm were normal and a grade II systolic pulmonic murmur in the second interspace to the left of the sternum was heard. Lungs were clear to auscultation and percussion except for diminished breath sounds over the left basal lung field. Abdominal examination showed a massive enlargement suggesting ascites with purplish pigmented striae over the left lower quadrant, hepatomegaly 3 fingerbreadths below the right costal margin, and splenomegaly 1 to 2 fingerbreadths below the left costal margin. There was moderate generalized abdominal tenderness, with rebound tenderness in the left upper quadrant, and normal peristalsis was heard. This prostate was enlarged to twice its normal size. The clinical impression was traumatic subcapsular hematoma incident to portal cirrhosis and hypertension with congestive splenomegaly.

Clinical studies on admission showed anemia, minimal hepatic dysfunction, and an enlarged splenic shadow on x-ray. Chest roentgenogram demonstrated evidence of a left basilar pneumonitis. Hemoglobin was 9.1 gm., hematocrit was 28, and leukocytes were 11,800 (table 1). Liver function studies (table 2) showed serum bilirubin, 1.6 mg. per 100 ml.; prothrombin time, 65 per cent; and thymol turbidity, 7 units. All other hepatic function studies, serum proteins, transaminase, and serum amylase were within normal limits. Accessory laboratory study results are given in table 3.

Scout films of the abdomen in the erect and recumbent positions demonstrated a soft tissue mass measuring 13 cm. in the left upper quadrant (fig. 1). Chest x-ray demonstrated increased lung markings over the left basal lung field. Bone marrow puncture studies were noncontributory. An electrocardiogram was normal.

On October 18, 1957, his temperature rose to 103° F., and he was sweating profusely. Rales could be heard at the base of the left lung. Evidence of

TABLE 1

Hematologic studies

	Date	Hemoglobin	Hematocrit	White Blood Cells	Segmented Cells	Nonsegmented Cells	Lymphocytes	Monocytes	Eosinophils	Basophils	Platelets	Bleeding Time	Coagulation Time
		gm.	%									*min.*	
Preoperative	10–18	7.9	22	9150	75	2	19	4					
	10–19	8.1	26	7250	76	2	13	7	2				
	10–20	8.8	29	9150	81	2	13	3	1				
	10–25	9.0	30	6400	73	0	24	1	2	0	144,000	8½	Silicon 41½ min.
	10–27	10.7	39	4650	66	4	21	7					3½ min.
	10–28	13.3	45	4250	74	3	18	4			172,000	5½	
Immediately postoperative	10–28	18.8									170,000		
Postoperative course		14	48	13,950							225,000	2	4 min. 35 sec.

TABLE 2

Liver function studies

	Serum Bilirubin	Van den Bergh Test	Bromsulphalein Retention	Alkaline Phosphate	Cephalin Flocculation	Thymol Turbidity	Prothrombin Time	Transaminase
	mg./100 ml.			*units*		*units*	%	*units*
Preoperative.....	1.6	Negative	All dye removed	1.83	2+	7	65	29
Postoperative.....	0.8	Negative		3.2	2+		100	75

ascites and peritonitis persisted. Hemoglobin was now 7.9 gm. and hematocrit 22 per cent. He was given 1000 ml. of whole blood. Surgical intervention was deferred in view of the patient's general condition. He was kept at absolute bed rest and given nothing by mouth; vital signs were watched closely. After 3 days of supportive therapy, evidence of diffuse acute peritoneal irritation had subsided and only tenderness and rebound tenderness in the left upper quadrant of the abdomen and a positive Kehr's sign remained. Pneumonitis had subsided and he was afebrile.

Serial scout films of the abdomen on October 21, 22, and 23 (fig. 2) revealed progressive enlargement of the spleen. An upper gastrointestinal x-ray demonstrated no evidence of gastric or duodenal ulceration or esophageal varices. The stomach was displaced to the right and slightly forward by the mass in the left upper quadrant. Loops of jejunum were compressed downward and medially (fig. 3). Anemia, thrombocytopenia and prolonged bleeding time persisted. It was necessary to give 3500 ml. of whole blood during the interval from October 24 through October 27.

Under general endotracheal anesthesia and with the patient in the supine position, a left rectus muscle splitting incision was made extending from the left costochondral junction to 5 cm. below the umbilicus. The abdominal cavity was filled with dark clotted and bright red blood. The liver was enlarged, hobnail in appearance, and consistent with Laennec's cirrhosis. Portal hypertension was evidenced by the extensive collateral venous channels. The portal vein was approximately four times normal size. The massive spleen filled the en-

TABLE 3

Accessory laboratory studies

	Serum Protein	Albumin	Globulin	Serum Amylase	Serum Lipase	Serum Ca^{++}	PO_4	Fasting Blood Sugar
	total	*gm.*	*gm.*	*Somogyi units*	*unit*	*mEq.*	*mEq.*	*mg.*
Preoperative.....	6.5	4.5	2	80				195
Postoperative.....	6.57	4.27	2.30	80	0.86	4.7	2.6	

FIG. 1. Soft tissue mass in the left upper and lower quadrant of the abdomen.

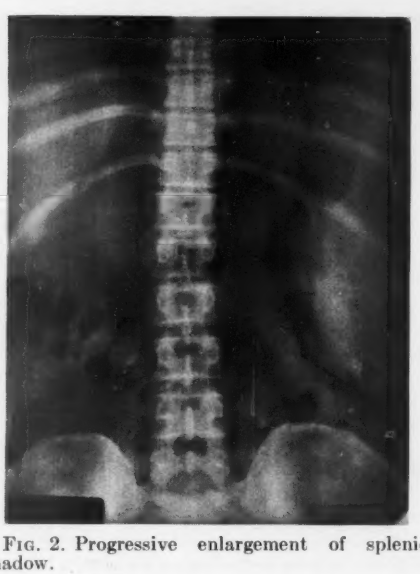

FIG. 2. Progressive enlargement of splenic shadow.

tire left upper and left lower quadrants of the abdomen. Neither liver biopsy nor portal pressures were taken because of the patient's precarious condition.

Attempts to deliver the spleen into the wound while still exerting digital pressure on the pedicle were unsuccessful. With blunt and sharp dissection the gastrosplenic, phrenocolic, and lienorenal ligaments were divided. With long right angle clamps the splenic artery, vein, and their branches were doubly clamped and with a clamp close to the hilus. The splenic pedicle was divided avoiding the tail of the pancreas. Large, warm gauze packs were placed in the retroperitoneal space and the spleen was delivered through the wound. A large subscapular tear with a large hematoma measuring 15

FIG. 3. Upper gastrointestinal x-rays demonstrating displacement of the stomach and small intestines.

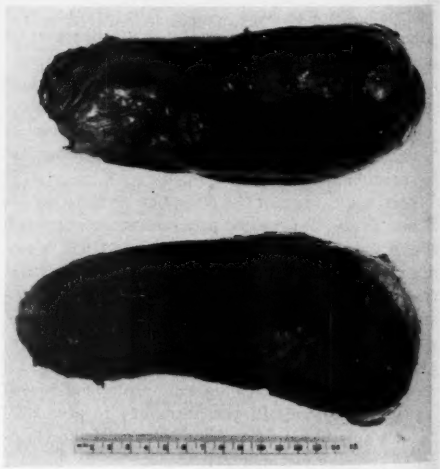

FIG. 4. Ruptured spleen with subcapsular tear.

by 5 cm. was found involving the posterior-inferior aspect of the spleen and its inferior pole (fig. 4). A large Penrose drain was placed in the left upper retroperitoneal space and brought out through a stab wound. Approximately 3000 ml. of serosanguineous fluid was aspirated from the abdomen; it was estimated that 2000 ml. of this was blood. The incision was closed in layers, by means of a continuous suture of no. 0 chromic catgut for the peritoneum, no. 30 cotton interrupted sutures for the fascial layers, no. 28 wire stay sutures through the anterior layer of the rectus sheath, and no. 60 interrupted cotton sutures for the skin.

During the surgical procedure the patient received 2000 ml. of whole blood, 40 ml. of 10 per cent calcium gluconate, and 700 ml. of 5 per cent glucose in water intravenously. The surgical specimen weighed 1450 gm.

During the immediate postoperative period the hemoglobin was 18.8 gm., and blood platelets 170,000. Nasogastric suction with a Levin tube was continued for 3 days. Approximately 2000 ml. of intravenous 10 per cent glucose in water were given daily. General supportive measures included chloromycetin, 500 mg., and streptomycin, 500 mg., twice daily for 10 days. The left flank drain was advanced over a 3-day period and removed on the 7th postoperative day. Sutures were removed on the 12th postoperative day. The patient made an uneventful recovery and was discharged on November 14, 1957, 17 days following operation.

COMMENT

Penetrating and nonpenetrating wounds of the abdomen have frequently been implicated in traumatic, spontaneous, or delayed rupture of the spleen. Automobile injuries or falls have been most frequently indicted. In our case a blow to the abdomen produced delayed rupture. This type of trauma accounted for 17.9 per cent of cases reported by Knopp and Harkins.[6] Rupture may be spontaneous due to laceration of the spleen or a considerable time may elapse between the initial trauma and secondary bleeding, in which case it is termed delayed hemorrhage. During the latent period, which may vary from several hours to 3 months, patients may be asymptomatic or have vague abdominal pain. With delayed hemorrhage following single or multiple lacerations of the spleen, a hematoma may form and bleeding be temporarily controlled. Depending upon the site of injury, the diaphragm or surrounding viscera may act as a tamponade. Byrne[4] reported delayed hemorrhage in 26 per cent of 101 patients; 21 patients bled within 7 days following injury and 1 patient after 14 days.

In the majority of cases studied by McIndoe,[7] Puestow,[9] and Rousselot and Illyne,[10] the interval from time of injury to delayed rupture varied from 2 to 21 days. Zabinski and Harkins,[14] in a study of 111 cases, found delayed rupture occurring in 5 to 40 per cent. The latent period was less than 14 days in 75 per cent of the cases. The splenic lacerations encountered were more commonly multiple. This was true of 22 per cent of Byrne's[4] cases, and 15 per cent were single lacerations. Rupture of this viscus occurs more commonly in men in the third and fourth decade of life.

Delayed rupture and splenic bleeding are dangerous. With a history of trauma, abdominal pain and tenderness, rigidity in the left upper quadrant, a positive Kehr's sign (pain referred to the left shoulder), a positive Ballance sign (shifting dullness in the right flank and fixed dullness in the left flank),[1] or associated rib fracture, a clinical diagnosis of splenic rupture can be made. Roentgenographic evidence of elevation of the left diaphragm, increased density in the left upper quadrant and enlarged splenic shadow with or without serrations along the greater curvature of the stomach (fig. 5), free fluid between intestinal loops, and displacements of the stomach or loops

FIG. 5. Enlarged splenic shadow with serrations along the greater curvature.

of small intestines medialward and downward, is diagnostic of spontaneous or delayed rupture.[8, 11, 13] Serial blood counts, hematocrit studies, and aspiration of free blood from the peritoneal cavity are of diagnostic significance.

If progressive hemorrhage occurs, early and adequate replacement with whole blood is necessary.

The triad of abdominal rigidity and tenderness and pain in the left shoulder were present in 34 per cent of the cases reported by Byrne.[4] Knopp and Harkins[6] found left upper quadrant tenderness in 43 per cent of 28 cases and shock and generalized abdominal tenderness in 4 per cent. Needle aspiration for blood in the peritoneal cavity was present in 6 of 8 cases reported by Knopp and Harkins. Others[5] have found this procedure useful, but a negative tap does not preclude the diagnosis of acute or delayed ruptures of the spleen. In our case the signs of peritoneal irritation especially confined to the left upper quadrant of the abdomen and a positive Kehr's sign were present. Serial hematocrit and roentgenographic surveys established the diagnosis of delayed rupture but operation was deferred because of the patient's poor condition. Following adequate blood replacement a splenectomy was done. Splenectomy is the treatment of choice for a ruptured spleen. McIndoe[7] reported 8 deaths in 9 unoperated cases. Zabinski and Harkins[14] state that the mortality in unoperated cases is 77 to 100 per cent. Scott and Bowman's[12] mortality figures were 46 per cent. Early diagnosis and immediate operation showed a decrease in the mortality rate. The range of operative mortality is 10 to 33 per cent.[3, 7, 14]

SUMMARY

1. Delayed rupture of the spleen in a 40-year-old man with portal cirrhosis, portal hypertension, and congestive splenomegaly, who sustained a blow to the left upper abdomen and developed a subcapsular hematoma, is reported.

2. Liver function studies were not consistent with the hepatic dysfunction usually accompanying this degree of portal cirrhosis.

3. The history of trauma, generalized abdominal pain, tenderness, and rebound tenderness in the left upper quadrant, a palpable spleen, and a positive Kehr's sign were diagnostic of subcapsular rupture of the spleen.

4. Serial scout films of the abdomen demonstrating splenic enlargement, elevation the left leaflet of the diaphragm and compression of adjacent viscera, and hematologic surveys revealing anemia were of importance in establishing the diagnosis and assessing the progress of the subcapsular rupture.

5. Splenectomy was successfully performed 14 days following trauma.

6. Splenectomy is the treatment of choice in all cases of subcapsular hematoma with spontaneous or delayed rupture of the spleen.

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REFERENCES

1. Ballance, C. A.: Quoted in 14.
2. Blain, A., III: The diagnosis and emergency management of ruptured spleen. Alexander Blain Hosp. Bull., *11:* 19, 1952.
3. Bueermann, W. H.: The latent period and delayed hemorrhage following traumatic rupture of the spleen. U. S. Naval M. Bull., *41:* 73, 1943.
4. Byrne, R. V.: Splenectomy for traumatic rupture with intra-abdominal hemmorhage; report of 101 cases. A. M. A. Arch. Surg., *61:* 273, 1950.
5. Byrne, R. V.: Rupture of spleen. Surg. Gynec. & Obst., *93:* 247, 1951.
6. Knopp, L. M., and Harkins, H. N.: Traumatic rupture of the normal spleen. Surgery, *35:* 493, 1954.
7. McIndoe, A. H.: Delayed hemorrhage following traumatic rupture of the spleen. Brit. J. Surg., *20:* 249, 1932.
8. O'Neill, J. F., and Rousseau, J. P.: Roentgenologic examination of the abdomen as an aid in the early diagnosis of splenic injury. Ann. Surg., *121:* 111, 1945.
9. Puestow, C. B.: Traumatic rupture of the spleen with delayed hemorrhage. S. Clin. North America, *20:* 195, 1940.
10. Rousselot, L. M., and Illyne, C. A.: Traumatic rupture of the spleen, with consideration of early fractures and late sequelae in 17 cases. S. Clin. North America, *21:* 455, 1941.
11. Sands, W. W.: The survey roentgenograms as an aid in the diagnosis of acute abdominal conditions. Surg. Gynec. & Obst., *97:* 4, 1953.
12. Scott, H. W., Jr., and Bowman, J. R.: Traumatic rupture of the spleen in childhood. J. A. M. A., *130:* 270, 1946.
13. Webb, R. C.: Traumatic rupture of the normal spleen with delayed hemorrhage. Lancet, *59:* 545, 1939.
14. Zabinski, E. J., and Harkins, H. N.: Delayed splenic rupture: a clinical syndrome following trauma. A. M. A. Arch. Surg., *46:* 186, 1943.

ECTOPIC BREAST TISSUE: REPORT OF UNUSUAL CASE*

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Polythelia, polymastia, hypermastia, or supernumerary breast tissue, whichever term one may choose to use, has always attracted attention. The formation of such breast tissue in the region of the breast proper and the adjacent axillary region is a rather frequent occurrence. The incidence of its occurrence varies up to 6 per cent. Arey[1] states that at least 1 per cent of a large population may show traces of supernumerary nipples. A conservative estimate of from 1 to 2 per cent would seem to be more exact. Accessory breast tissue occurs in equal numbers in both sexes.

Supernumerary breasts are unilateral or bilateral, and if multiple, usually occur in the milk line.[4] These masses are frequently diagnosed as lipomas and appear as soft, lobulated, fatty tumors enclosing a small core of breast tissue. The diagnosis of breast tissue should be strongly considered if the patient gives a history of these masses having occurred at puberty coincident with the growth of the normal breast, if they enlarged at preganancy, or if they underwent cyclic changes coincident with the menstrual period.

The relatively rare observation of benign or malignant tumor formation in these frequently occurring anomalies gives the impression that they are essentially harmless. Breast tissue which maintains its physiologic function and is located in the axillary region may require excision. Carcinoma occurring in congenitally displaced breast tissue carries a poor prognosis. This is usually due to the proximity of the regional lymphatics.

From an embryologic viewpoint, supernumerary breast tissue may be found at any point along the mammary ridge or milk line which runs from the axilla to/or through the lateral border of the pubic region to end on the upper medial surface of the thigh. The presence of breast tissue in some parts of the vulva is explainable on this developmental basis.[5]

The milk line is first noted during the sixth week of development and consists of two bandlike structures of ectoderm.[9] The level at which the breasts will develop is dependent on the species. As a general rule, the breasts in the human are confined to a relatively small area in the pectoral region but in certain instances can occur anywhere along the entire course of the previously described milk line. The different levels in the human race at which the breast occurs in racially different groups is probably an indication of the utilization of slightly different levels of the milk line in their development. The human breast begins as a localized thickening of the milk line in the region of the location of the future breast. By the eighth week of embryologic development, it is difficult to recognize the milk line in its entirety.[7] In the region where the future breast is to be located, the masses of epithelial cells extend deep into the mesenchyme. Growth in the breast system is very slow. At birth there is little difference between the breast tissue of the male and female. In the male the breast structures seldom develop appreciably from the infantile state. Likewise, in the female there is little structural change until puberty, when the areola becomes elevated through a deposition of fat, and then there follows rapid enlargement of the breasts, which assume a conical shape with the areola and nipple at the apex. In regard to the selective development of the milk line in the human, all but the cranial one-third of the ridge disappears and, from this portion, the normal breast develops.

McFarland[8] states the first reported case of breast tissue in the vulva was that of Hortung.[6] He described a mass the size of a goose egg attached to the left labia majoria by a pedicle the size of a man's thumb in a 30-year-old woman. It was congenital in origin, enlarged at puberty, manifested changes with the menstrual period, and exuded milky fluid from an eroded area which, on careful examination, revealed a retracted nipple. The treatment instituted in this case was not recorded.

De Blasio,[3] in 1905, reported the case of a young woman who had bilateral masses of breast tissue attached to each labium the size of a hen's egg and each had a well formed nipple. In this patient, only the nipples were present at puberty,

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with the remainder of the gland developing later. The maximum size of the masses was reached during pregnancy.

Bell,[2] in 1926, reported the case of a 23-year-old woman who, while in her third pregnancy, fell upon a sharp object and sustained an injury in the region of her vulva. Soon thereafter, a yellowish-white fluid exuded from the region of the injury and a mass appeared. With each succeeding pregnancy, of which there were nine, the mass increased in size, and became smaller during periods when the patient was not pregnant. When this patient was seen at the age of 59 years, there was a vulvar mass the size of a hen's egg attached by a pedicle approximately 3 cm. in diameter. This mass was described as being variable in consistency, quite elastic, and without attachment to the overlying skin. At operation, the tumor measured 7 by 5 cm., and there was no evidence of a nipple. Microscopic examination of the tissue was reported as "resting breast with cystic disease present."

McFarland,[8] in 1931, reported the case of a 34-year-old colored woman who, at 6 months' gestation during her third pregnancy, was found to have a pendulous, rounded mass attached to the lower one-third of her right labium by a short, wide base. The overlying skin was smooth and without hair, and the tumor mass was somewhat tender to touch. It was stated that an exact history could not be elicited owing to the mental status of the patient, but according to her, this mass had not been present with any of her previous pregnancies. A tentative diagnosis of fibroma of the vulva was made and the tumor was excised. At operation, it was 5 by 3 by 2.5 cm. and covered by deeply pigmented skin. Beneath the skin there was an encapsulated node so loosely attached to the surrounding tissue that it appeared to be separate. The tumor was lobulated, and, on cut section, was coarsely granular, porous, and pale yellow in color, alternating with pinkish-white stroma. Photomicrographs of this tissue and sections from a normal breast at the same period of gestation were so similar that the tissue had to be labeled to prevent confusion of the two.

Both fibroadenoma and carcinoma have been reported to develop in aberrant breast tissue occurring in the vulva. Since a carcinoma in this area carries such a poor prognosis owing to the proximity of large and superficial lymphatic chains, it would seem justifiable to remove such masses at the earliest possible time.

In reviewing the reported cases of breast tissue located in the vulva, it is obvious that it is not a frequent occurrence. While the finding of such tissue in the vulva unilaterally is not uncommon, a careful review of the literature reveals only three reported instances of patients in which breast tissue occurred bilaterally. The case which we report herewith is thought to be the fourth such instance in which breast tissue has been found bilaterally in the vulva.

CASE REPORT

M. K., a 26-year-old colored woman, gravida III, para III, abortus 0, was first seen in the Vanderbilt University Hostpial Clinic on September 29, 1954 ,with the complaint of "my breasts are busting" (fig. 1). She gave a history of swelling of her breasts for several days prior to the clinic visit. There had been no discharge from the nipples and there was no history of trauma. With cessation of the last menstrual period there had been no decrease in the size of the mammary glands. She also described a burning sensation throughout both breasts. Her last born infant had been breast fed, and had been weaned 6 months prior to the clinic visit. Her last menstrual period, which had ended 2 days previously, had been of 3 days' duration. The menstrual history was not remarkable otherwise.

Her physical examination on the first visit revealed multiple large, firm, nodular areas in both breasts. The breasts were enlarged and pendulous. In the lateral portion of the right breast there was an area in which the overlying skin was shiny and resembled orange peel in appearance. No secretions could be expressed from the nipple of the breast. There were no palpable nodes in the axilla. Pelvic examination at this time revealed a cysto-

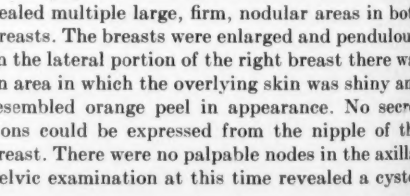

FIG. 1. Note hypertrophied breasts, for which initial treatment was sought. Breasts weighed a total of 28 lb. 13 oz. when removed.

cele and rectocele. She was treated with support to the breasts and application of heat at night.

On follow-up visit 1 week later, the breasts seemed to be somewhat improved, with less swelling and tenderness. She was to continue per prescribed treatment. Two weeks later the orange peel appearance was still present and, in addition, two masses 3 by 4 cm. were noted in the medial quadrant of the right breast.

On October 26, 1954, the patient was admitted to Vanderbilt University Hospital for a biopsy of the breast lesions. At that time the breasts were described as being pendulous, and the right breast was considerably larger than the left. In the lower portion of the right breast the nipple was retracted. This was thought to be due to the edema surrounding the nipple. Just superior to the nipple there was an area of erythema with tenderness and an orange peel appearance. Underlying this area of erythema a mass 4 by 6 cm. was present. The left breast was described as normal and without nipple discharge. On pelvic examination at this time the vulva was described as being normal. The impression was inflammatory carcinoma of the right breast.

On the day of admission an attempt was made to aspirate the mass in the breast, which was thought by some who examined the patient to be a galactocele. The attempt was unsuccessful, but the operator made the statement that on entering the mass with the aspirating needle he encountered some resistance. Chest film on admission was normal. Eight days later, under general anesthesia, the mass in the breast was biopsied. Pathologic examination revealed mastitis without evidence of carcinoma. The patient was discharged on the 1st postoperative day, November 3, 1954, to be seen in the postoperative clinic 5 days later.

Approximately 6 weeks later she returned to the surgery clinic at Vanderbilt Hospital, and stated that 1 month prior to this visit she had noticed a small mass in the medial inferior portion of the right breast. Occasionally there was a burning and stinging sensation in this area. The mass had increased in size and the night before coming to the clinic it had begun to drain. The drainage was described as being watery in consistency. The patient was of the opinion that her breast had increased in size. On examination the right breast was somewhat larger than the left and both were warm and displayed the orange peel phenomenon. A mass 2.5 by 2.5 cm. was noted in the medial inferior quadrant of the right breast. An ulcerated area draining a clear, watery fluid was present. The draining material was of small volume and the area was not painful. No axillary lymphadenopathy was present. Pelvic examination at this time revealed a uterus the size of 3 months' gestation, and a Hogben test was positive. No mention was made of the appearance of the vulva on this examination.

On February 2, 1955, a bilateral simple mastectomy was done. The total weight of both breasts was 28 lb. 13 oz. Each breast measured 30 cm. in diameter. Her hospital course was uneventful. She was followed in the obstetric clinic until August 20, 1955, at which time she was delivered of a normal 6-lb. male infant.

The patient was admitted to the Nasville General Hospital on August 16, 1956. Physical examination revealed a pregnant uterus of approximately 8 months' gestation, and bilateral vulvar masses which were of such size that it was felt they would obstruct the vaginal outlet at the time of delivery. In addition, there was a mass in the left axillary region. The vulvar masses were firm and rubbery in consistency, freely movable, and attached by broad bases. The overlying skin was smooth and shiny. There was no regional lymphadenopathy. The axillary mass was likewise firm, rubbery in consistency, and freely movable. No axillary nodes were palpable.

FIG. 2. Patient M. K. approximately 2 years following bilateral simple mastectomy. Note the aberrant breast tissue in the left axilla.

FIG. 3. Masses of breast tissue occurring in the labia during third trimester of pregnancy. The right labial mass measured 8 by 7 by 4 cm., and the left 15 by 8 by 4.5 cm.

The presumptive diagnosis at the time of admission was that the axillary mass was hyperplasia of a remnant of unremoved breast tissue or accessory breast tissue, and the vulvar masses were due to massive edema. One examiner who saw the patient in the Nashville General Hospital Tumor Clinic mentioned the fact that these tumors might be breast tissue (figs. 2, 3).

On August 29, 1956, the patient was taken to the operating room, and under gas, oxygen and ether anesthesia, the two vulvar masses were excised. On gross examination they had the appearance of breast tissue.

The tumor from the left labium consisted of three masses of tissue, the largest of which was 8 by 7 by 4 cm. The tumor from the right labium measured 15 by 8 by 4.5 cm. The report of microscopic sections was as follows:

"There are lobules of highly secretory breast tissue, the lobules of which are well circumscribed. Intervening between the lobules is moderately dense fibrous connective tissue which shows some fibroblastic proliferation and slight infiltration by chronic inflammatory cells. The tissue contains numerous dilated ducts, some of which are filled with pink staining fluid. A few glands lined by acidophilic epithelium like that of the apocrine sweat glands are scattered here and there. Diagnosis: accessory secretory breasts of vulva, bilateral."

The patient remained in the hospital and on September 5, 1956, went into labor spontaneously and was delivered of a 7-lb. 9-oz. male infant. Her hospital course was uneventful except for some separation of the vulvar wound edges which subsequently healed without difficulty. She was discharged from the hospital on September 13, 1956. When seen in the clinic several weeks later the axillary mass had decreased in size considerably and the vulvar incisions were completely healed.

SUMMARY

Ectopic breast tissue may occur at any location along the milk line extending from the axilla to the medial surface of the upper thigh. The incidence of its occurrence is from 1 to 2 per cent and such tissue may be either unilateral or bilateral. Due to the rare occurrence of malignant change, these masses give the impression of being relatively harmless. When malignant changes do occur, the prognosis is poor owing to the proximity of regional lymphatics.

Breast tissue occurring in the vulva unilaterally, though reportedly rare, is not too uncommon. A careful review of the literature reveals only 3 reported cases in which breast tissue was found in the vulva bilaterally. The fourth such case is reported here.

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REFERENCES

1. Arey, L. B.: *Developmental Anatomy*. W. B. Saunders Company, Philadelphia, 1954.
2. Bell, J. W.: Supernumerary breast near labium. Am. J. Obst. & Gynec., *11:* 507, 1926.
3. De Blasio, A.: Polimastia penivulvare. Arch. di Psichiat., *26:* 171, 1905.
4. DeCholnoky, T.: Supernumerary breasts. Arch. Surg., *39:* 926, 1939.
5. Fisher, J. H.: Fibroadenoma of supernumerary mammary gland tissue in vulva. Am. J. Obst. & Gynec., *53:* 335, 1947.
6. Hartung, E.: Inaugural Dissertation, Erlangen, 1872.
7. Maximow, A., and Bloom, W.: *A Textbook of Histology*, Ed. 6. W. B. Saunders Company, Philadelphia, 1952.
8. McFarland, J.: Mammary gland situated on the labium majus. Arch. Path., *11:* 236, 1931.
9. Patten, B. M.: *Embryology of the Pig*, Ed. 3. Blakiston Company, Philadelphia, 1948.

SUCCESSFUL CARDIAC RESUSCITATION IN THE RECOVERY ROOM*

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Cardiac resuscitation by means of an open thoracotomy is not a new procedure, the first case having been reported in 1902.[10] In almost all of the reported successful cases, the procedure was done in a surgical operating room where the patient was under the close scrutiny of an anesthetist and a surgical team, and where the necessary sterile supplies and instruments were instantly available.

In recent years several cases of successful resuscitation outside the operating room have been reported. Reagan and his associates,[7] and Beck and co-workers[1] have reported cases treated in the emergency room of a hospital. Mozen,[6] Celio,[4] Turk,[11] and Schwartz[8] and their respective associates reported successful cardiac resuscitation of patients who were in hospital rooms or open wards. Brown and co-workers[2] reported a successful case treated with a penknife thoracotomy in an x-ray darkroom. Southworth and his associates[9] reported a successful defibrillation in a patient undergoing cardiac catheterization. Other successful resuscitations outside the operating room have been reported by Hamel and co-workers[5] and Bücherl and his associates.[3]

This is a report of a cardiac arrest successfully resuscitated in the recovery room after extensive pelvic surgery. The reporting of these cases is urged so that the alert physician is encouraged to attempt to save a patient in cardiac arrest regardless of the equipment at hand. The recent extension of the scope of the procedure outside the operating room makes successful resuscitation possible if the physician acts quickly and decisively.

CASE REPORT

Mr. V. F., a 62-year-old white man, entered this hospital on June 22, 1957, with complaints of gross hematuria, dysuria, and frequent urination of several months' duration. Six months prior to this admission a segmental resection of the bladder for an infiltrating carcinoma had been performed at another hospital.

Physical examination revealed a thin, chronically ill patient who appeared older than his stated age. The chest had an emphysematous configuration, and occasional wheezes were heard on auscultation. Bimanual examination of the pelvis revealed a 6-cm. mass involving the base of the prostate and the floor of the bladder and extending to the right lateral wall of the pelvis. This mass was hard, nontender, and mobile.

Intravenous pyelography showed good function and delineation of the left upper urinary tract. There was delayed function on the right side with moderate dilation of the renal pelvis and ureter. A chest roentgenogram showed mild emphysema, and an electrocardiogram revealed a normal sinus rhythm with a right bundle branch block. The hemoglobin was 12.2 gm., and the blood urea nitrogen was 15 mg. per 100 ml. Cystoscopy revealed an extensive tumor of the bladder involving primarily the right lateral wall and trigone. Because of the symptomatology and the obstruction of the right upper urinary tract, we decided to perform a cystectomy and urinary diversion.

On July 26, 1957, cystectomy, ureterosigmoidostomy, and proximal colostomy were performed through an abdominoperineal approach. During the 6-hour procedure the patient received 5500 cc. of blood. He had no hypotensive or anoxic episodes and was sent to the recovery room in good condition.

When the patient arrived in the recovery room the blood pressure was 60/40, the pulse rate was 60, and respiration was labored. A moment later there were no blood pressure, pulse, audible heart sounds, or breath sounds. Pounding on the chest caused no response. Without the usual sterile precautions, an incision was quickly made through the left fifth intercostal space. The heart was found in standstill.

Within 4 minutes of the onset of cardiac arrest, rhythmic cardiac compression was begun. Simultaneously, tracheal intubation was attempted by the anesthesiologist. The attempt proved unsuccessful, thus necessitating a tracheotomy through which positive pressure oxygen was administered. While the tracheotomy was being performed, the airway was maintained with oxygen supplied through a face mask. Shortly after cardiac massage was begun it was noted that the heart was in fibrillation. Massage was continued for 45 minutes. At that point the heart was defibrillated with two

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series of electric stimuli. The heart then resumed its normal sinus rhythm with a blood pressure of 90/60. The changes in cardiac rhythm were recorded with a continuous electrocardiogram. Blood pressure was maintained by a slow intravenous drip of Neosynephrine. This was discontinued after 3 hours. Spontaneous respiration returned about 1 hour after cardiac resuscitation was begun.

The heart was observed for 15 minutes, then the chest was closed. A Foley catheter attached to underwater suction was used for intrapleural drainage. The patient responded to questioning shortly after spontaneous respiration started. He could move all his extremities, and there were no signs of neurologic damage.

The urinary output was only 250 cc. the 1st postoperative day, but rose to 700 cc. on the 2nd day and remained normal thereafter. The blood urea nitrogen rose to a maximum of 136 mg. per 100 ml. on the 9th day, but slowly declined and was within normal limits 1 month later. A postoperative intravenous pyelogram showed normal upper urinary tracts. During the 1st postoperative week the patient developed a pulmonary acidosis which responded to frequent tracheal aspiration, Alevaire and Isuprel nebulizer, and intermittent oxygenation of the tracheotomy. On the 14th postoperative day the tracheotomy tube was removed.

The thoracotomy wound healed *per primum*, but the patient had a persistent suprapubic urinary fistula. The patient left the hospital on October 29, 1957, for a leave of absence of 1 month. The patient was completely clear mentally and had no signs of neurologic impairment. He returned to the hospital on December 4, 1957, for closure of the urinary fistula. An attempt to repair the fistula under local anesthesia was unsuccessful. The patient died of inanition on December 29, 1957, 5 months after successful cardiac resuscition. Permission for autopsy was not granted.

COMMENT

In this case, the combination of factors responsible for the revival of the patient were the presence of the operating team at the time of cardiac arrest, the prompt detection of cessation of vital signs, and the immediate decision to perform thoracotomy. The importance of close observation of the patient in the recovery room until vital signs have stabilized cannot be overemphasized. Although the combination of circumstances resulting in successful resuscitation in these cases is admittedly rare, the increasing number of recent reports offers hope for future extension of the procedure outside the operating room.

The essential steps in cardiac resuscitation are: (1) thoracotomy through the fourth or fifth intercostal space and rhythmic cardiac compression; (2) establishment of an effective airway by means of an anesthesia machine with an endotracheal tube in place or, if this is not available, direct mouth-to-mouth breathing until the proper equipment is at hand; and (3) restoration of the spontaneous rhythmic heartbeat with an electric pacemaker or defibrillator.

Emergency equipment, including a thoracotomy set, anesthesia apparatus, and plasma expanders, should be available at key areas in the hospital such as the emergency room, recovery room, and each ward floor. Frequent briefing of the attending and house staffs on the techniques involved will increase their awareness of the problem, impress them with the necessity for immediate decisive action, and overcome the trepidation inherent in any situation requiring such drastic intervention.

SUMMARY

A case of successful cardiac resuscitation in the recovery room following a total cystectomy is presented. The salient points in the management of cardiac arrest outside the operating room are discussed.

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REFERENCES

1. Beck, C. S., Weckesser, E. C., and Barry, F. M.: Fatal heart attack and successful defibrillation: new concepts in coronary artery disease. J. A. M. A., *161:* 434, 1956.
2. Brown, C. D., Knudson, J., and Schroeder, G. F.: Cardiac arrest at work: penknife thoractomy with recovery. J. A. M. A., *163:* 352, 1957.
3. Bücherl, and Koch, R.: Erfolgreich behandelter Herzstillstand trotz verspätet einsetzender Herzmassage, Thoraxchirurgie, *4:* 261, 1956.
4. Celio, A.: Cardiac arrest associated with coronary occlusion: successful resuscitation. J. Internat. Coll. Surgeons, *25:* 299, 1956.
5. Hamel, N. C., and Brewer, L. A., III: Cardiac arrest one hour following pneumonectomy. A. M. A. Arch. Surg., *75:* 277, 1957.
6. Mozen, H. E., Katzman, R., and Martin, J. W.: Successful defibrillation of heart:

resuscitative procedure started on medical ward and completed in operating room. J. A. M. A., *162:* 111, 1956.

7. Reagan, L. B., Young, K. R., and Nicholson, J. W.: Ventricular defibrillation in patient with probable acute coronary occlusion. Surgery, *39:* 482, 1956.
8. Schwartz, A. M., and Lobell, S. M.: Cardiac arrest: successful treatment in a patient's room. J. A. M. A., *166:* 488, 1958.
9. Southworth, J. L., McKusick, V. A., Peirce, E. C., II, and Rawson, F. L., Jr.: Ventricular fibrillation precipitated by cardiac catheterization: complete recovery of patient after 45 minutes. J. A. M. A., *143:* 717, 1950.
10. Starling, E. A., and Lane, W. A.: Report of society of anesthetists. Lancet, *2:* 1397, 1902.
11. Turk, L. N., III, and Glenn, W. W. L.: Cardiac arrest: results of attempted cardiac resuscitation in 42 cases. New England J. Med., *251:* 795, 1954.

THE CARCINOID NEOPLASM AND SYNDROME: A REVIEW WITH EXTENSION BY UNUSUAL CASES*

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Tumors arising from the argentaffin cells of Kulchitsky have demanded clinical attention and gained their niche in the literature. In 1955, Waldenstrom and his colleagues[5] brilliantly correlated the pathologic behavior of this neoplasm with various clinical syndromes. Thus emerged the "carcinoid syndrome," metastatic carcinoid neoplasm liberating serotonin to cause pulmonic stenosis, asthma, cutaneous flushing, abdominal cramps, and diarrhea. The formation of osteoblastic metastases in 1 case and the development of the syndrome in a 12-year-old girl have expanded our knowledge of this neoplasm. These 2 unusual cases stimulated a review of carcinoid neoplasms at the University of Cincinnati Hospitals. During the last 10 years, 50 cases were admitted and will be discussed with these 2 unusual case reports.

CASE REPORT

Case 1

History of present illness. A 47-year-old white man was admitted to the surgical service on May 8, 1957, complaining of nausea, vomiting, and diarrhea of 6 days' duration. Weakness 2 days prior to admission forced him to bed. On the day of admission he noted mild right lower quadrant pain.

Three years previously he commenced having attacks similar to the present one—episodes of nausea, vomiting, diarrhea, and weakness lasting for several days. Each attack was accompanied by flushing of his face, shoulders, and chest. The patient had had a laparotomy for a suspected acute cholecystitis at another hospital in 1955, at which time numerous tumor nodules were found in the liver and one or two in the omentum. Exploration of both the large and small bowel revealed no primary source for the tumor. A liver biopsy was reported as metastatic carcinoma. On this admission a rounded infiltrate was noted in the left midlung field and was thought to represent either primary tumor or a metastatic lesion. Following the operation the patient grew worse, and he had had recurrences of the previously described attacks every 2 or 3 weeks. He had lost 20 to 30 lb. since that time. He had been able to continue his work, however, as a night watchman. He had been a heavy drinker until 1955.

Physical examination. His temperature was 100.2° F., blood pressure 80/70, and pulse 120. On admission this middle aged, white man demonstrated an erythematous flush of the face, neck, upper arms, and chest with a mild cyanosis about the lips and the tip of the nose. There was a maculopapular dermatitis over this area which extended down the back to the buttocks. There was a severe hyperemic conjunctivitis and pharyngitis. The skin was cool and moist. Auscultation of the chest revealed a few rales in the left base. The abdomen was rigid but demonstrated no tenderness and normal bowel sounds were heard. The liver was palpated 8 cm. below the right costal margin. There was a healed right upper abdominal incision. The lateral lobes of the prostate gland were enlarged, the left being more prominent.

Laboratory. A hemogram revealed hemoglobin, 15.8 gm. per 100 ml., and the white blood count, 26,600. Differential diagnosis revealed neutrophils, 89 per cent; lymphocytes, 11 per cent; hematocrit, 55 per cent; and blood urea nitrogen, 74 mg. per 100 ml. Blood chlorides were 100 mg. per 100 ml., and CO_2 was 16.2 mg. per 100 ml. Serum amylase was 80 units. Urine analysis, including porphobilinogen, was negative. Blood sodium and potassium were 137.5 and 4.5 mEq., respectively. The spinal fluid revealed no cells and a normal amount of protein. Blood and stool cultures were negative. An electrocardiogram was normal. Liver profile and Bromsulphalein retention were normal. The total protein was 7.00 gm. per 100 ml. with an albumin to globulin ratio of 3.55:4.45, or 0.80. Blood calcium and phosphorus were 8.2 and 7.7 mg. per 100 ml., respectively.

Roentgenograms. A chest roentgenogram revealed a left, midlung field nodule, 4 cm. in size, probably neoplastic, with probable sclerosing lesions of the ribs. An abdominal roentgenogram revealed extensive hepatomegaly with cholelithiasis and sclerosing lesions of both the ilia and

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FIG. 1. Admission roentgenogram showing carcinoid neoplastic metastases to the lung and rib.

FIG. 2. Roentgenogram revealing osteoblastic metastases to vertebrae and pelvis.

sacroiliac joints and of the eighth, ninth, and twelfth vertebrae (figs. 1 and 2).

Course in the hospital. The above symptoms, characteristic flush, and 2-year history of surprising work activity following discovery of metastatic tumor in the liver, suggested the diagnosis of metastatic carcinoid. As most carcinoid bone metastases are either microscopic or osteolytic, the radiologic determination of osteoblastic metastases cast doubt upon the clinical diagnosis.

On conservative therapy with intravenous electrolytes and plasma, the hypotension and tachycardia were corrected. The urinary output of 30 cc. for the first 24 hours became 1500 cc. by the 3rd day. In spite of this and a later diuresis, the blood urea nitrogen rose to 136 mg. per 100 ml., and the potassium rose to 6.3 mEq. on May 12, 1956, supporting the diagnosis of azotemia from lower nephron nephrosis secondary to hypotension from the excessive loss of gastroenteric fluid.

On the 2nd day a urine test was strongly positive for serotonin (5-hydroxyindole acetic acid) (5-HIAA). The positive strength of the test and the fact that it was positive through July 12 negate the effect of his preadmission small doses of Thorazine.

After 2 days the patient was much improved; the diffuse erythema had faded, and the abdomen became soft. Within 10 days he was afebrile, eating well, and asymptomatic. The diuresis reached 7 to 8 L. of urine a day as his blood chemistry returned to normal and the patient became convalescent.

Further investigation was then pursued. Prostatic biopsy was normal. Cardiac catheterization on May 21, 1957, was entirely normal and serotonin levels on the blood drawn from the right ventricle were normal. Barium enema, gastrointestinal, and small bowel series were negative.

Pathology report. A bone biopsy of the right posterior iliac spine revealed carcinoid tumor (figs. 3, 4, and 5). The slides from the previous laparotomy were reviewed and the diagnosis of carcinoid, identical to the above biopsy, was made.

Carcinoid metastases to bone are usually microscopic, as seen in figure 4; with growth of metastases, osteolytic bone destruction is usually seen.

Clinic note. On August 22, 1957, this patient was seen in the outpatient department, at which time he was doing well and was free of gastrointestinal symptoms. He stated, however, that he still had occasional flushes. There was moderate erythema of the face at that time. No cardiac murmur was heard. No abdominal masses were palpable other than an enlarged liver.

Comment. The flushing, vomiting, diarrhea, and reasonable well being supported the presumptive diagnosis of metastatic carcinoid. The roentgenogram showing osteoblastic bone metastases was disconcerting, as osteoblastic metastases are extremely rare. The bone biopsy, its similarity to the liver biopsy taken in 1955, and the high urinary 5-HIAA content established the diagnosis. In our series of the last 10 years, 13 of the 48 carcinoid

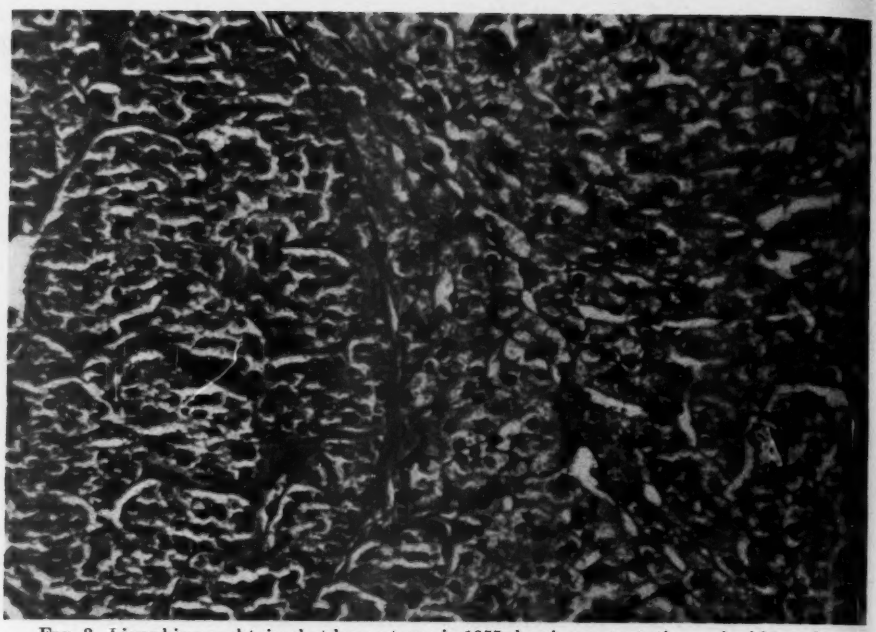

FIG. 3. Liver biopsy obtained at laparotomy in 1955 showing metastatic carcinoid neoplasm.

FIG. 4. Needle aspiration biopsy of pelvic bone metastasis

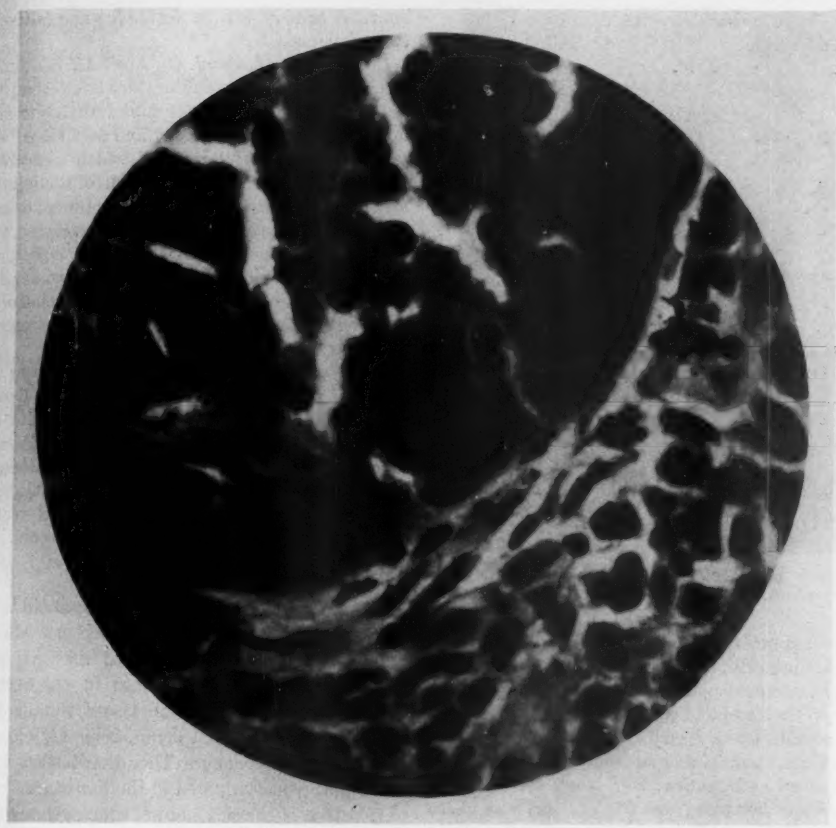

FIG. 5. Needle aspiration biopsy of pelvic bone metastasis

neoplasms had metastisized, but none to bone. Eldred[1] and MacDonald[2] have reported finding microscopic bone marrow invasion by carcinoid neoplasm—the usual finding when the bone is involved. Waldenstrom[4, 5] described a carcinoid-like tumor that caused osteoblastic metastases similar to those in this patient. He classified this tumor as adenocarcinoma. He also mentioned 3 other cases of carcinoid tumor with bone metastases reported by others.

The explanation of the abdominal rigidity is uncertain. Mills[3] relieved tetany in a patient during an acute syndrome by intravenous calcium gluconate. In this patient the rigidity was confined to the abdominal musculature and was not relieved by calcium gluconate. During his azotemia, with a blood urea nitrogen of 136 mg. per 100 ml., the blood calcium was 7.1 mg. per 100 ml. and the phosphorous was 10 mg. per 100 ml.

The blood albumin was low, with the albumin-globulin ratio being 3.55:4.45, or 0.8. These values were obtained 2 days after his acute symptoms and their significance is unknown. The existence of radiopaque gallstones and the premature advanced calcification of his vessels suggested a metabolic defect, which is the subject of further study (fig. 6).

Oliguria and azotemia are not commonly seen during the acute syndrome. Serotonin reduces the renal blood supply and increases tubular absorption, giving an antidiuretic effect. The fluid and electrolyte loss with vomiting and diarrhea and the hypotensive effect of serotonin can explain the lower nephron nephrosis.

Case 2

History of present illness. This 12-year-old white girl was studied for obestity. On examination an intense mottled redness of the face was seen. The mother stated that this first appeared when the patient was 8 years old. In the last 4 years she had

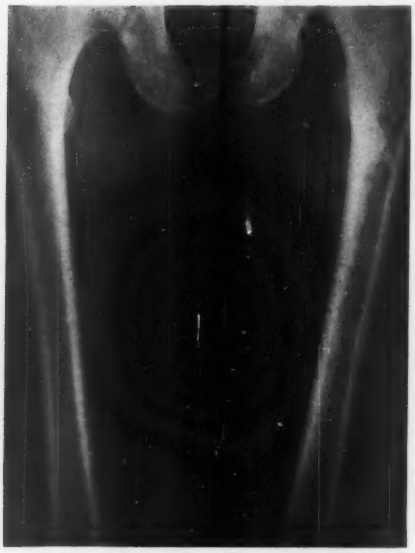

FIG. 6. Roentgenogram of legs showing arterial calcification.

occasional attacks of vomiting, diarrhea, and dizziness. Formerly, these symptoms were thought to be an emotional reaction. The increasing awareness of the carcinoid syndrome and the positive serotonin test established the diagnosis of the carcinoid neoplasm, and laparotomy was advised.

Physical examination. Her blood pressure was 120/85 and her pulse was 96. The girl was obese with a redness of the face, arms, and legs. A grade I harsh pulmonic systolic murmur was heard. The liver was palpable 2 fingerbreadths below the right costal margin.

Laboratory results. Urinalysis revealed a specific gravity of 1.013, pH of 5.5, and 15 to 20 red blood cells per high power field (menstruating). A hemogram revealed a red blood count of 4.25 million with 13.3 gm. of hemoglobin and a white count of 9200 with 41 lymphocytes, 4 monocytes, and 2 basophils. Two stools were negative for ova, parasites, and blood, and one stool was positive for occult blood.

An upper gastrointestinal series as well as a small bowel study and a barium enema were all interpreted as being within normal limits. Bromsulphalein retention was 1.5 per cent; phosphorous was 3.6 mg. per 100 ml.; alkaline phosphatase was 6.8 units; bilirubin was 0.18 mg. per 100 ml., direct, and 0.58 mg. per 100 ml., total; and thymol turbidity was 1.9 units. Serum proteins were: albumin, 4.65 gm. per 100 ml., and globulin, 2.25 gm. per 100 ml. The albumin-globulin ratio was 2.06. Cephalin flocculation was negative in 24 and 48 hours. A urinary test for 5-HIAA was positive on two occasions.

Course in the hospital. On April 27, 1957, under general anesthesia, an exploratory laparotomy was performed. The liver was found to be tremendously enlarged, both right and left lobes, with tumor nodules, the largest of which measured 2 inches in diameter. At the junction of the jejunum and ileum a stenosing lesion of the ileum, extending for a distance of 2 cm. and involving two-thirds of the circumference of the bowel, was found. There was extension of this tumor into the root of the mesentery. A wedge resection of the area (without removing the base of the mesentery) and liver biopsy were performed.

Pathology report. The tissue diagnosis was carcinoid of the ileum with metastases to the liver.

Comment. The usual age of patients with carcinoid tumors as given in the literature is 55 years. In our series the average age was 61 years when the tumor was benign and 64 years when malignant. This 12-year-old patient with a typical picture alerts us to the prevalence of this syndrome.

DISCUSSION

In the last 10 years, 48 cases of carcinoid tumor were seen in the University of Cincinnati Medical Center. Eleven were appendiceal in origin, 34 in the small bowel, and 3 arose in the rectum. Thirty-one of the 34 small bowel tumors were located in the terminal ileum, 2 in the jejunum, and 1 in the duodenum This distribution varies from that usually quoted in the literature.

Thirteen of the small bowel tumors were malignant. Nine were diagnosed at laparotomy and were treated surgically. Four were found incidentally at autopsy. The only other malignant carcinoid arose in the rectum. Follow-up of the 9 malignant tumors treated surgically shows only 2 survivors. The average survival time from the

TABLE 1

Symptoms of 13 cases of malignant carcinoid neoplasm

Symptoms	Number	Per Cent
Vasomotor	3	23
Abdominal—diarrhea, borborygmi, hyperperistalsis, cramping pains, etc.	9	69
Valvular heart lesions	0	0
Asthma	0	0
Arthritis	0	0
Emotional disturbances	1*	8

* Psychosis.

onset of symptoms is 8.2 years and from the time of diagnosis, 3.5 years.

Seldom does a case have the entire gamut of symptoms that are possible in this syndrome. Table 1 illustrates the incidence of symptoms encountered in this series: nine cases presented merely abdominal symptoms and 3 presented vasomotor manifestations. There were no cases with arthritis, asthma, or heart lesions.

SUMMARY

1. Two unusual cases of carcinoid syndrome are presented; 1 in a young girl, and 1 with osteoblastic metastases.

2. Carcinoid tumors treated at the University of Cincinnati Medical Center are reviewed.

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REFERENCES

1. Eldred, W. J.: Argentaffin carcinomas. A. M. A. Arch. Surg., *73:* 517, 1956.
2. MacDonald, R. A.: A study of 356 carcinomas of the gastrointestinal tract. Report of four new cases of the carcinoid syndrome. Am. J. Med., *21:* 867, 1956.
3. Mills, G. Y.: The syndrome of intestinal carcinoid, pulmonary valvular stenosis and cutaneous flush. Ann. Int. Med., *45:* 1213, 1956.
4. Waldenström, J., Pernow, B., and Silwer, H.: Case of metastasizing carcinoma (argentaffinoma?) of unknown origin showing peculiar red flushing and increased amounts of histamine and 5-hydroxytryptamine in blood and urine. Acta med. scandinav., *156* 73, 1956–1957.
5. Waldenström, J., and Ljungberg, E.: Studies on the functional circulatory influence from metastasizing carcinoid (argentaffine, enterochromaffine) tumors and their possible relation to enteramine production. Acta med. scandinav., *152:* 293, 1955.

STAINLESS STEEL MESH REPLACEMENT OF ANTERIOR ABDOMINAL WALL; RADICAL EXCISION OF RECURRENT DESMOID TUMOR*

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Repair of the anterior abdominal wall in the presence of marked tissue deficit is a problem that every surgeon faces at times. This is most frequently encountered in the repair of large ventral hernias, as reported by Koontz,[15] Lam and his associates,[17] Preston,[25] and Throckmorton,[29] but it sometimes is a challenge in the resection of extensive tumors. Resection of malignant tumors with massive involvement is usually palliative, as noted by Anderson and Witkowski.[1] However, even extensive desmoid tumors can be cured by adequate resection, particularly when done as the primary procedure, if the difficulty of closure does not deter the surgeon from a sufficiently radical procedure.

The unusual characteristics of desmoid tumors make this possible, since they are potentially malignant but never metastasize. A radical approach is warranted and may be curative. Such a case is presented, even though the outcome was unsuccessful, to emphasize the availability of a method of abdominal wall closure simple enough to encourage adequate surgery initially in all cases of desmoid tumor of the anterior abdominal wall.

Desmoid tumors are hard, slow growing, unencapsulated, infiltrating fibromas of musculofascial origin. Regional lymph node and distant metastases never occur, but 25 per cent[10, 21] to 31 per cent[23] tend to recur after surgical excision, owing to incomplete removal of the characteristic infiltration of tumor cells into surrounding muscle, indicating a certain degree of malignancy. Although no metastases have been observed, malignant transformation has been described by Pack and his associates,[4, 21] Donald and Caylor,[9] and Green.[11]

The etiology of desmoid tumors is unknown. It has been related to sexual physiology by reason of their predominance in females and their tendency to develop during or shortly after pregnancy. Of 598 cases collected from seven reports,[10, 18, 20-23, 28] 85 per cent were females; 88 per cent of these females had had a more or less recent pregnancy. Geschickter and Lewis[10] and Pfeiffer[23] noted that growth seems more rapid during gestation, and recurrences appear to Pack and Ehrlich[21] to be precipitated by pregnancy with uncommon frequency. A high concentration of gonadotropic substance was found in such a tumor by Geschickter and Lewis.[10] Traumatic origin is suggested by their developing in surgical scars, at the site of injury, and as a result of the physiologic trauma of gestation and parturition.

Treatment of choice is unequivocally early, wide surgical resection beyond the apparent cellular infiltration. Adequate resection at the initial procedure, when opportunity is most favorable, will produce a permanent cure since metastases never occur. Mason,[18] Nichols,[20] Pearman and Mayo,[22] and Stewart and Mouat[28] felt that radiation is beneficial only from a palliative standpoint. The efficacy of a hormonal therapeutic approach needs further investigation.

Wide resection of extensive tumors may produce abdominal wall defects that are extremely difficult to close. The largest desmoid tumor on record is the 17-kg. tumor removed by Rokitansky,[27] with closure of more than the peritoneum and skin being impossible. Following radical excision of these tumors, fascia lata was used by Bessesen[3] and omental graft by Cattell and Wiedman[7] for repair. Recurrent desmoid tumors particularly become extensive. Ye and his associates[30] completely excised an extensive recurrent desmoid tumor, tantalum mesh and complicated plastic techniques being required for closure. Booher and Pack[4] reported the most invasive desmoid tumor in the literature, with mesenteric involvement precluding resection. The intestinal tract was not invaded, nor was evidence of such invasion found in perusing 38 reports on desmoid tumors since 1880, although peritoneal infiltration is quite common.

Adequate resection without regard to closure is essential for cure. This was recommended as

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long ago as 1904 and 1905 by Pfeiffer[23] and Powers.[24] Modern surgical techniques have greatly simplified closure as well as diminished the morbidity and mortality rates, so that recurrence rates should accordingly decrease. Closure of large defects has been accomplished successfully, securely, and relatively simply by implantation of an artificial prosthesis.[5, 6, 19, 26] Metallic mesh prostheses are readily available in unlimited quantities, with no operative trauma, and have the advantages—but none of the disadvantages—of autogenous materials, according to Guy and his associates[12] and Lam and his associates.[17] Survey of the literature revealed that the 12- by 12-inch piece of tantalum mesh used by Koontz[15] was the largest prosthesis for replacement of a defect of the abdominal wall previously reported. Tantalum and stainless steel mesh cause no significant tissue reaction, even when used to replace a deficiency of the peritoneum, according to the investigations of Crile and King[8] and Koontz.[15] They provide adequate support through their inherent strength and that of the fibrous tissue which enmeshes them. Review of the literature[2, 13, 14, 16, 25] and clinical experience indicate that tantalum and stainless steel are quite comparable, with no particular advantages of one over the other.

CASE REPORT

This 24-year-old, white, married woman was admitted to St. Luke's Hospital on February 4, 1957. Following an appendectomy in 1949, a desmoid tumor developed in the region of the operative scar and was removed in 1950. Wide excision of a recurrence with tantalum mesh replacement of the resulting muscular defect was accomplished in 1952. Pregnancy terminating in a normal delivery immediately followed the excision of a second recurrence in 1953.

Examination on this admission revealed multiple tumors of the anterior abdominal wall, although multiple tumors have been noted in only a few previous cases (by Pack and Ehrlich[21] and Stewart and Mouat[28]). A large, hard, irregular, 10- by 20-cm. mass was palpated under the well healed scar which extended from just below the right costal margin to the pubis. The left upper and lower quadrants each contained similar masses about half this size. Smaller tumors were scattered through the full length of the abdomen near the midline as well as caudal to the large mass in the right lower quadrant.

Radical excision was performed on February 13.

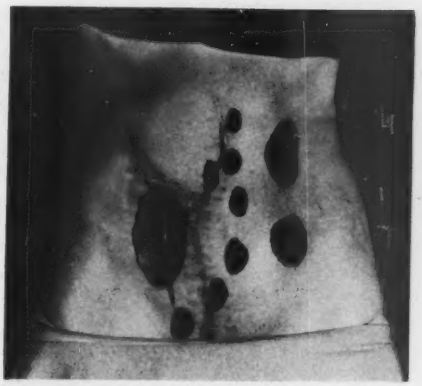

FIG. 1. The distribution of the multiple anterior abdominal wall tumors is indicated as well as the location of the old operative scar.

A midline incision was made from the xiphoid to the pubis. Wide undermining of the skin to the posterior axillary line on the right and the anterior axillary line on the left provided adequate exposure. Exploration of the abdomen revealed extensive invasion of the right lower quadrant tumor into the mesentery of the distal ileum and right colon and into the ascending colon itself. The distal 60 cm. of the ileum and the entire right colon were excised *en bloc* with the whole right half of the anterior abdominal wall except for the skin. The tumor was adherent to the duodenum, but there was no apparent invasion of this structure; adherence to the right iliac crest was also noted. Portions of the serosa of the duodenum and of the periosteum of the right iliac crest were removed. Ileotransverse colostomy was accomplished. The remaining tumors were limited to the abdominal wall, the left half of which was excised slightly less radically, leaving the peritoneum and skin. Bilateral oophorectomy was performed by Dr. George Finola. The limits of the resection of the anterior abdominal musculature were the lateral costal margins cephalad, the pubis caudad, the anterior axillary line on the left, and the posterior axillary line at the lateral border of the latissimus dorsi on the right. Resection of the peritoneum was necessarily so extensive that only partial closure could be accomplished.

A 30- by 40-cm. stainless steel mesh replacement prosthesis was implanted on the peritoneum, consisting of three sheets of 50-0.003, 18-80 alloy mesh sutured to each other with continuous steel wire. The loops of bowel which could not be covered with peritoneum were thus in contact with the mesh. The mesh was attached with interrupted steel sutures to the limits of resection of musculo-

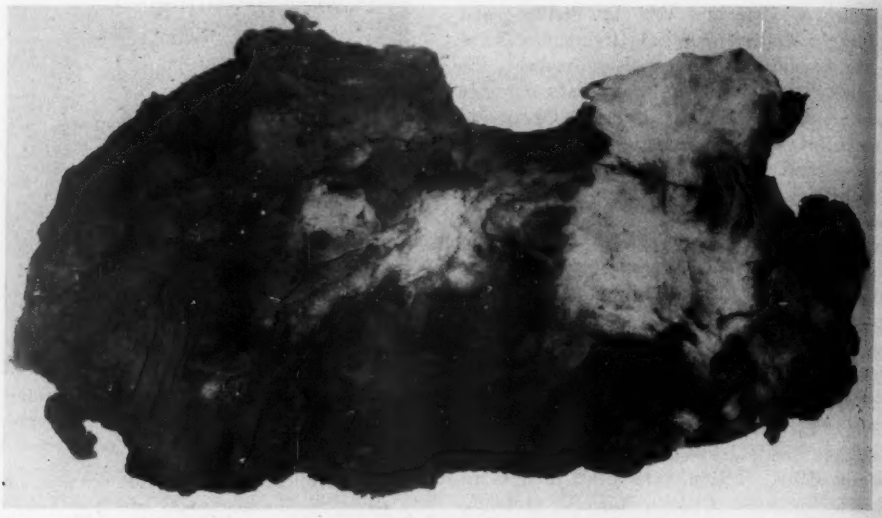

FIG. 2. This portion of the surgical specimen shows the tumor in the musculature above extending downward through the mesentery and into the wall of the ascending colon.

fascial layers of the abdominal wall. The large, thin skin flaps were closed without tension over the mesh after removal of a portion of the right skin margin containing half of the old scar. Moderate serous drainage without infection was drained from the dead spaces anterior and posterior to the mesh through soft rubber tubes with intermittent suction. Healing was quite uneventful; a 2-cm. slough of skin at the junction of the two scars closed by secondary intention in spite of the exposed mesh.

Pathologic examination revealed actual invasion of the tumor into the wall of the ascending colon, as well as into its mesentery and that of the terminal ileum. All of the hard, tough fibrous masses were similar in gross and microscopic appearance. Poorly to moderately cellular, dense, fibrillar, fibrous tissues were seen microscopically, with mitoses present among the spindle shaped cells.

She was followed closely, and no signs of recurrence of the abdominal wall tumors developed. In March 1958, 13 months after surgery, an intra-abdominal tumor was palpated in the right side of the abdomen. Intravenous pyelogram revealed a rather marked right hydronephrosis and hydroureter. Upper gastrointestinal x-rays showed the stomach to be pushed upward and to the left. Barium enema was negative except for the well functioning ileotransverse colostomy.

Abdominal exploration was performed on May 7, 1958. The abdomen was opened by resecting the

FIG. 3. This x-ray, taken 4 months postoperatively, revealed the mesh to be intact, although only the margins and seams are visible in the photograph. The extent of the mesh is apparent, but, by this time, the soft tissues to which the mesh was attached had stretched sufficiently to make the prosthesis appear 5 cm. smaller in each dimension than when implanted.

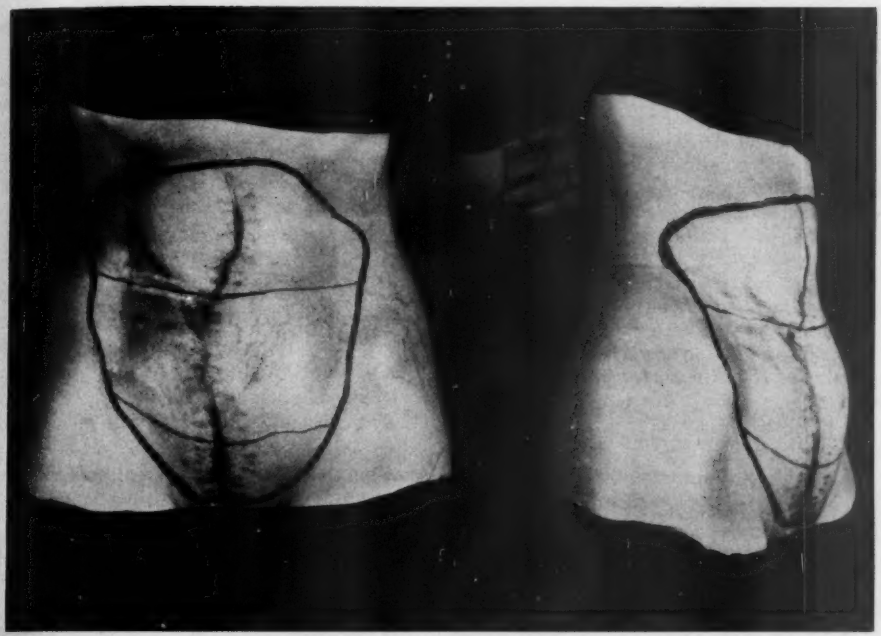

FIG. 4. The palpable margins and seams of the mesh are marked. The surgical scars are visible. The adequate abdominal support provided by the mesh 9 months postoperatively is demonstrated.

previous skin scar and incising the mesh vertically. The intraabdominal tumor was freely movable and was attached locally to the mesh in the right upper quadrant, so that this quadrant of the prosthesis was removed. It was then found that two distinct but connected tumor masses involved the entire mesentery of the small bowel. Resection would have demanded a duodenotransverse colostomy in the second portion of the duodenum and was not accomplished. Right nephrectomy was done. Again the peritoneum was left open. The resected portion of the prosthesis was replaced with two sheets of stainless steel mesh in a similar manner. The divided mesh was repaired readily with stainless steel wire sutures.

Although it was discouraging to have such a massive recurrence after the previous radical surgery, this procedure did provide an opportunity to examine the mesh after it had been in place for 15 months. The stainless steel was intact with no fragmentation. Fibrous tissue firmly enmeshed the wire screen. Both surfaces were covered with a smooth sheet of tissue, with apparent complete replacement of the deficient peritoneum. There were few adhesions between the mesh and the underlying exposed bowel. The overlying skin was freely movable.

The patient recovered from this surgery uneventfully and is again asymptomatic. The mesh provides adequate abdominal support, but, of course, the patient has no voluntary contraction of the anterior abdominal wall. Posture is quite good and all movements are accomplished well except for the patient's inability to sit up from the supine position without using her arms.

It is of interest that she had no complaints referable to her abdomen on either admission, the first being precipitated by disabling pain in her lower back, radiating into both legs but more severe on the right. Episodes of such pain had occurred intermittently at about monthly intervals since the patient was 6 years of age. This pain improved on physical therapy preoperatively, but it is noteworthy that it has not recurred since the radical operation although no obvious cause was demonstrated. Prior to this admission she had planned to have multiple subcutaneous tumors of the scalp, neck, back, chest and all four extremities removed for cosmetic purposes. These had been developing for years and 25 such tumors were present, ranging in size from 0.2 to 4 cm. in diameter. Excision biopsy of five of these was done to rule out metastases. All were epidermal retention cysts. The abdominal wall tumors were

naturally given priority, but she recovered sufficiently from the initial surgery to have the other 20 retention cysts plus five new ones removed in June 1957.

Scarring, palpated at the rectosigmoid junction on exploration of the abdomen initially, was investigated postoperatively. Proctoscopy revealed 14 small rectal polyps, which were removed. There was no evidence of malignancy, and repeated proctoscopic and barium enemas have revealed no other lesions.

It is also interesting that two subcutaneous tumors of the scalp and one involving the entire left half of the hyoid bone were excised from her 3-year-old son in November 1956. Pathologic examination of all three tumors revealed only scar tissue.

DISCUSSION

A more extensive resection than has previously been reported for a desmoid tumor was accomplished in this case. There had been three recurrences, multiple tumors were present in the abdominal wall, and there was more intraperitoneal involvement than has previously been described, including the first reported involvement of the gastrointestinal tract itself. Still there were no metastases and a cure was possible with adequately extensive surgery. Had a comparable radical excision been performed initially, a permanent cure would have almost undoubtedly resulted. As it was, surgery was sufficiently successful to prevent local recurrences in the anterior abdominal wall; it is believed that the tumor which is still present arose from the inadequately resected posterior abdominal wall.

Closure required the implantation of a larger replacement prosthesis for the anterior abdominal wall than has been described heretofore. This was accomplished with ease; one need have no fear regarding closure when such extensive resection of abdominal musculature and peritoneum is indicated. Radical resection as accomplished offered the only possibility of permanent cure. After such extensive surgery, the relatively simple insertion of an artificial replacement prosthesis was the only feasible method of closing the massive defect involving the full thickness of the abdominal wall except for the skin. Replacement of almost the entire anterior abdominal wall with stainless steel mesh has proved highly satisfactory to date.

This tumor originally arose in a surgical scar, but pregnancy probably stimulated growth in the recurrence. Therefore, and because the prosthetic abdominal wall obviated gestation from a mechanical standpoint, further pregnancies were contraindicated. Sterilization by bilateral oophorectomy was elected to remove any possible estrogenic influence on this tumor, as suggested by Geschickter and Lewis[10] and Pack and Ehrlich.[21]

SUMMARY

Repair of the anterior abdominal wall in spite of massive tissue deficiency can be accomplished successfully with relative simplicity by implantation of an artificial prosthesis.

Desmoid tumors are locally invasive and potentially malignant, with a high rate of recurrence after excision, but they never metastasize. Local excision that is sufficiently radical to assure complete removal will result in permanent cure. It is imperative that these tumors are approached in this manner at the initial procedure, and difficulty of closure need no longer deter one from performing adequate surgery.

A case is presented in which the third recurrence of a desmoid tumor, which arose initially in an appendectomy scar, was apparently precipitated or at least aggravated by pregnancy. Multiple isolated tumors, which are unusual, extensively involved the anterior abdominal wall, and the largest invaded the ileocolic mesentery and the right colon itself. It is to be noted that intraperitoneal involvement is rare, and invasion of the gastrointestinal tract has not been previously reported. Wide radical excision of all tumor tissue was accomplished, including almost the entire anterior abdominal wall except for the skin. All of the musculofascial elements and part of the peritoneum of the abdominal wall were successfully replaced by the largest metallic mesh prosthesis reported to date. Bilateral oophorectomy was performed to avoid further pregnancies and to obviate estrogenic stimulation of the tumor, since there appears to be a definite etiologic relationship to sexual physiology and trauma.

Recurrent tumor again developed 13 months later, and abdominal exploration revealed that resection was not feasible. The stainless steel mesh was in excellent condition 15 months after implantation, performing its function well. The divided mesh was readily repaired, and the resected portion of mesh was easily replaced. If

such a radical excision had been performed initially, a permanent cure could have been expected with confidence. Recurrences are much less likely to be completely amenable to excision, so that the conservative approach has no place in the treatment of desmoid tumors. It is again emphasized that radical surgery at the initial procedure is mandatory. Any defect of the abdominal wall which might result from radical excision can be readily and securely repaired with an artificial replacement prosthesis of metallic mesh.

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REFERENCES

1. Anderson, R. E., and Witkowski, L. J.: Use of tantalum mesh for difficult abdominal closure. Am. Surgeon, *23:* 139, 1957.
2. Babcock, W. W.: Range of usefulness of commercial stainless steel cloths in general and special forms of surgical practice. Ann. West. Med. & Surg., *6:* 15, 1952.
3. Bessesen, D. H.: Desmoid tumor of abdominal wall. Am. J. Surg., *16:* 513, 1932.
4. Booher, R. J., and Pack, G. T.: Desmomas of abdominal wall in children. Cancer, *4:* 1052, 1951.
5. Brown, A. B., and Koontz, A. R.: Tantalum mesh repair of ventral hernia followed by term pregnancy and delivery. Am. J. Obst. & Gynec., *70:* 1364, 1955.
6. Bussabarger, R. A., Dumouchel, M. L., and Ivy, W. H.: Use of tantalum mesh to repair a large surgical defect in the anterior abdominal wall. J. A. M. A., *142:* 984, 1950.
7. Cattell, R. B., and Wiedman, J. G.: Desmoid tumors. Lahey Clinic Bull., *8:* 2, 1952.
8. Crile, G., Jr., and King, D. E.: Successful use of tantalum mesh to repair abdominal wall defect in presence of massive fecal contamination. Surgery, *29:* 914, 1951.
9. Donald, J. M., and Caylor, H. D.: Fibrosarcoma developing in a desmoid. Ann. Surg., *89:* 631, 1929.
10. Geschickter, C. F., and Lewis, D.: Tumors of connective tissue. Am. J. Cancer, *25:* 630, 1935.
11. Green, C. C.: Desmoid tumor. Arch. Surg., *50:* 304, 1945.
12. Guy, C C., Werelius, C. Y., and Bell, L. B., Jr.: Five years experience with tantalum mesh in hernia repair. S. Clin. North America, *35:* 175, 1955.
13. Jonas, K. C.: Stainless steel cloth as an internal prosthesis. Arch. Surg., *60:* 1205, 1950.
14. Koontz, A. R., and Kimberly, R. C.: Tissue reactions to tantalum mesh and wire. Ann. Surg., *131:* 666, 1950.
15. Koontz, A. R.: Difficult hernias: use of tantalum mesh in repair. J. Internat. Coll. Surgeons, *16:* 637, 1951.
16. Koontz, A. R., and Kimberly, R. C.: Tissue reactions to tantalum mesh and stainless steel gauze: an experimental comparison. Ann. Surg., *137:* 833, 1953.
17. Lam, C. R., Szilagyi, D. E., and Puppendahl, M.: Tantalum gauze in repair of large post-operative ventral hernias. Arch. Surg., *57:* 234, 1948.
18. Mason, J. B.: Desmoid tumors. Ann Surg., *92:* 444, 1930.
19. Narat, J. K., and Khedroo, L. G.: Repair of abdominal wall defects with fortisan fabric. Ann. Surg., *136:* 272, 1952.
20. Nichols, R. W.: Desmoid tumors: report of 31 cases. Arch. Surg., *7:* 227, 1923.
21. Pack, G. T., and Ehrlich, H. E.: Neoplasms of the anterior abdominal wall with special consideration of desmoid tumors. Internat. Abstr. Surg., *79:* 177, 1944.
22. Pearman, R. O., and Mayo, C. W.: Desmoid tumors. Ann. Surg., *115:* 114, 1942.
23. Pfeiffer, C.: Die Desmoide der Bauchdecken und ihre Prognose. Beitr. klin. Chir., *44:* 334, 1904.
24. Powers, C. A.: Fibroid growths of abdominal wall. J. A. M. A., *45:* 676, 1905.
25. Preston, D. J.: Repair of abdominal hernia with steel cloth implant. J. Internat. Coll. Surgeons, *18:* 513, 1952.
26. Pridgen, J. E., and Tennison, C. W.: Reconstruction of entire anterior abdominal wall in presence of postradiation changes. Am. J. Surg., *92:* 54, 1956.
27. von Rokitansky, K.: Exstirpation eines Fibroms der vorderen Bauchwand. Wien. med. Presse, *21:* 105, 140, 1880.
28. Stewart, M. J., and Mouat, T. B.: Fibroma of abdominal wall. Brit. J. Surg., *12:* 355, 1924.
29. Throckmorton, T. D.: Tantalum gauze in repair of hernias complicated by tissue deficiencies. Surgery, *23:* 32, 1948.
30. Ye, R. C., Devine, K. D., and Kirklin, J. W.: Extensive recurrent desmoid tumor of abdominal wall: radical excision followed by reconstruction of abdominal wall with plastic procedure. Plas. & Reconstruct. Surg. *12:* 59, 1953.

A COMPARISON OF THE EFFICACY OF ANTIBIOTICS IN OPEN AND CLOSED LOOP STRANGULATION OBSTRUCTION*

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The existing high mortality rate in strangulation obstruction has stimulated a continuing interest in the pathophysiology and management of this condition.[8] A considerable amount of evidence has accumulated which indicates that bacteria play a significant if not dominant role in this not uncommon abdominal catastrophe.[2, 7] Following strangulation, the lumen of a segment of ileum has been shown to be the site of accumulation of large amounts of dark, foul fluid. With progression of intestinal necrosis the bowel wall becomes permeable to this material and it gains access to the peritoneal cavity. Peritoneal fluid resulting from strangulated bowel was found to be toxic for normal dogs in amounts as small as 3 cc. per kg.[3] When the altered segment of of bowel was placed in a plastic bag so that exposure to the animal was prevented, then life was prolonged.[5] It has been shown that neomycin and Chloromycetin will render the resulting fluid nontoxic when placed inside the lumen of the strangulated bowel.[1, 2] The survival rate of normal animals exposed to toxic strangulation obstruction fluid was considerably improved by the simultaneous administration of antibiotics. The larger the dose of antibiotic, the better were the animals' chances for survival.[4]

Blain and Kennedy[6] demonstrated the value of antibiotics in open loop strangulation obstruction. They also conducted experiments utilizing isolated jejunal segments. The purpose of the following experiments was to compare the relative efficacy of antibiotics in the treatment of open and closed loop strangulation obstruction. Fluid and electrolytes were not administered during these studies.

MATERIALS AND METHODS

Thirty-six adult mongrel dogs were used. Intravenous Nembutal was utilized for anesthesia in all cases. Sterile technique was observed during all operative procedures. The small intestine was exposed through a low midline abdominal incision. After completion of the operative procedure upon the intestine, the abdominal wound was closed with cotton sutures.

Series 1

A 10-cm. closed loop of obstructed ileum was created by passing umbilical tape ties around each extremity of the altered segment. The venous drainage from this area was then ligated. All marginal vessels were divided and ligated (fig. 1). The segment was returned to the abdomen which was closed in layers with cotton sutures. The animals were placed in their cages for observation of survival time.

Group 1. The 9 animals which composed this group were given penicillin (600,000 units) at the time of completion of the operative procedure and at 12-hour intervals thereafter.

Group 2. The 9 animals of this group received no antibiotics.

Series 2

The ileum near the cecum was obstructed by placing an umbilical tape tie around its entire circumference. The venous drainage to a 30-cm. loop of ileum proximal to the site of obstruction was divided and ligated. The marginal veins were treated in a similar manner (fig. 2). The abdominal wound was closed after the altered bowel had been replaced.

Group 1. Nine of the animals with open loop obstruction were given 600,000 units of penicillin after completion of the operative procedure. Similar doses were given at 12-hour intervals thereafter as long as the animals survived.

Group 2. Nine animals with open loop obstruction received no penicillin.

RESULTS

Series 1

Group 1. All animals survived for 24 hours but less than half were alive at 48 hours. The mean

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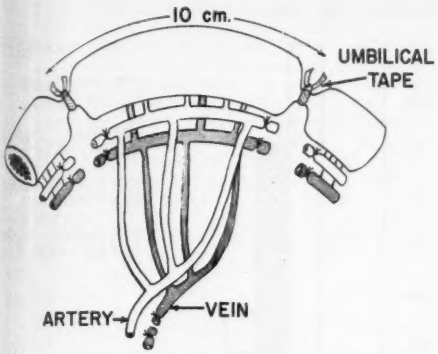

FIG. 1. Method of strangulating closed loop of ileum.

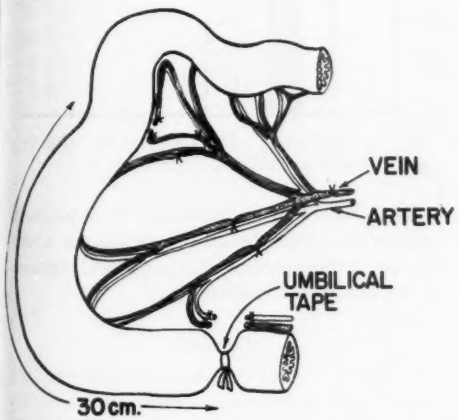

FIG. 2. Method used to strangulate open loop of obstructed bowel.

survival time was 55.5 hours (range 24 to 94 hours) (table 1).

Group 2. In this group of animals with closed obstruction which did not receive penicillin there were no 48-hour survivors. Two of these animals failed to survive for 24 hours. The mean survival time was 29.2 hours (range 18 to 42 hours) (table 2).

Series 2

Group 1. Where penicillin was given to animals with open loop obstruction there were 8 out of 9 dogs alive after 90 hours. The mean survival time was 109.8 hours (range 24 to 168 hours) (table 3).

Group 2. All animals with open loop obstruction were alive after 24 hours. The mean survival

TABLE 1

Results in animals with closed loop obstruction (penicillin given)

Dog Number	Survival Time
	hr.
205	40
20	36
80	46
100	36
40	24
200	91
27	65
58	68
212	94
Mean	55.5

TABLE 2

Results in animals with closed loop obstruction (no penicillin given)

Dog Number	Survival Time
	hr.
288	20
209	42
282	24
230	24
263	24
267	18
216	42
202	41
154	28
Mean	29.2

TABLE 3

Results in animals with open loop obstruction (penicillin given)

Dog Number	Survival Time
	hr.
112	168
283	93
285	116
90	90
247	118
174	118
204	93
202	168
203	24
Mean	109.8

TABLE 4

Results in animals with open loop obstruction (no penicillin given)

Dog Number	Survival Time
	hr.
224	71
236	117
234	44
239	150
206	36
204	53
225	120
27	120
269	72
Mean	87.0

time was 87.0 hours (range 36 to 150 hours) (table 4).

COMMENT

Following strangulation of an open loop obstructed bowel the lumen rapidly fills with blood and plasma elements. This material serves as an effective culture medium so that the bacterial flora normally found in the gut increases rapidly. Stasis of the bowel content resulting from obstruction also promotes bacterial proliferation. Compromise of the integrity of the altered gut wall is temporarily postponed because the toxic intraluminal fluid escapes into the proximal loop of bowel. The normal intestinal wall serves as an effective barrier to toxic strangulation fluid since harmful effects are seen only after the material reaches the peritoneal cavity. Eventually necrosis of the strangulated bowel wall progresses to the point of allowing the toxic material to escape into the peritoneal cavity, causing the death of the animal.

In closed loop strangulation obstruction the intraluminal fluid builds up great pressure because no route for decompression exists. This hastens permeability of the gut wall with earlier peritoneal exposure of the animals. These pathophysiologic differences probably account for the longer survival time as seen in open loop strangulation obstruction (fig. 3). Administration of penicillin to animals with open loop strangulation obstruction resulted in prolongation of life for an average of 23 hours. When this antibiotic was given to animals with closed loop obstruction the mean survival time was increased by 26 hours. It appears that the efficacy of penicillin is about the same in both groups after peritoneal exposure to the fluid has occurred.

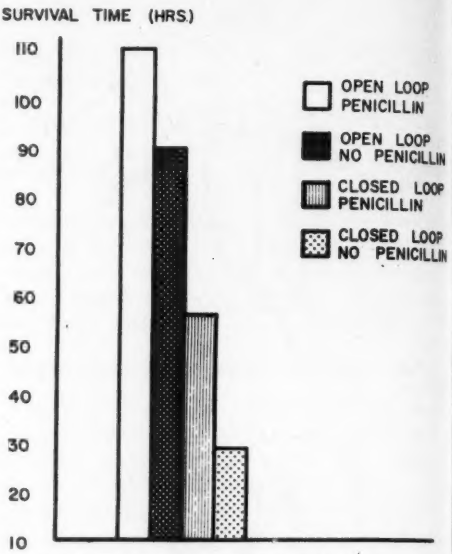

FIG. 3. The effect of antibiotics in open and closed loop strangulation obstruction.

SUMMARY

1. The mean survival time for animals with closed loop strangulation obstruction was 29 hours. This value was increased to 55.5 hours by the administration of penicillin.

2. The mean survival time for animals with open loop strangulation obstruction was 87 hours. Survival time for these animals was increased to 109.8 hours by the administration of penicillin.

3. The mean extension of survival time following administration of penicillin was about the same for both groups of animals.

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REFERENCES

1. BARNETT, W. O.: The efficacy of Chloromycetin in the treatment of strangulation obstruction, Ann. Surg., in press.
2. BARNETT, W. O., AND DOYLE, R. S.: The effects of neomycin upon the toxicity of peri-

toneal fluid resulting from strangulation obstruction. Surgery, *44:* 442, 1958.

3. Barnett, W. O., and Hardy, J. D.: Observations concerning the peritoneal fluid in experimental strangulated intestinal obstruction: the effects of removal from the peritoneal cavity. Surgery, *43:* 440, 1958.
4. Barnett, W. O., and Messina, A. J.: The influence of massive antibiotics in experimental strangulation obstruction. Gastroenterology, in press.
5. Barnett, W. O., and Stanley, T. W.: The treatment of experimental strangulation obstruction, Arch. Surg., *77:* 196, 1958.
6. Blain, A., III, and Kennedy, J. D.: The effect of penicillin in experimental intestinal obstruction; studies on strangulated low ileal obstruction. Bull. Johns Hopkins Hosp., *79:* 1, 1946.
7. Cohn, I., Jr.: Bacterial factors in strangulation obstruction: review of experimental work. Am. Surgeon, *22:* 836, 1956.
8. Nemir, P., Jr., Hawthorne, H. R., Cohn, I., Jr., and Drabkin, D. L.: The cause of death in strangulation obstruction: an experimental study. I. Clinical course, chemical, bacteriologic and spectrophotometric studies. Ann. Surg., *130:* 857, 1949.

TRACHEOSTOMY FOR RESPIRATORY MUSCLE FAILURE IN INFANTS AND CHILDREN*

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The indication for any tracheostomy is the need to establish or guarantee normal exchange of air in the alveoli of the lung for the absorption of oxygen and the elimination of carbon dioxide.[5] Tracheostomy is a well established procedure for high mechanical obstruction in infants and children. It is our purpose to call attention to a more subtle and insidious cause of life threatening respiratory failure in very young infants and children, where, under carefully controlled conditions, we feel that tracheostomy is also indicated. The reader must be strongly aware, however, that these cases should be very carefully selected, as tracheostomy in infants and children can be very harmful if the most careful technique and post-tracheostomy care are not followed. Emphasis is on the important effect of tracheostomy on respiratory physiology in this age gorup other than by relieving mechanical obstruction in or above the larynx. Carter and Giuseffi[4] demonstrated the value of tracheostomy in reducing the volume of the respiratory tract dead space and in decreasing the resistance to flow of air by circumventing the larynx. A third advantage is the ease of removing secretions. If we include these indications in carefully selected cases of respiratory insufficiency, some infants and children will be saved who otherwise might be lost. The beneficial effects of removing secretions from lower respiratory passages, reducing dead space, and diminishing resistance to air flow by tracheostomy have recently been observed in 3 infants and 1 small child. The procedure was believed to be a major contributing factor to their recovery.

EFFECTS OF ALTERED RESPIRATORY PHYSIOLOGY

Effect of drying secretions. Secretions in the respiratory tract are removed effectively by normal mechanisms. Ciliary activity and active cough reflex are the most important of these. Positional change, postural drainage, and recently, lung physiotherapy, and in acute cases, tracheobronchial toilet are important adjuvants. Ciliary activity is reduced by drying and thickening of mucus and by drugs and poisons. Diminution of ciliary activity, weakening of the cough and swallowing reflex, overproduction, and thickening of secretions all contribute to lower respiratory tract obstruction.

Effect of progressive loss of function of respiratory effort due to various causes. The strength of the respiratory muscles, the integrity of the nervous reflex pathways for their innervation, the conscious and subconscious, and the chemical and physical regulatory mechanisms which result in efficient respiration are complicated and often difficult to separate or identify separately. It has been shown by many studies that decreasing strength of any of these mechanisms by action of a wide variety of disease states, or by experimental laboratory methods, results in reduction of effective respiration which is usually progressive and insidious in its progress.

Secondary effect of hypoxia and additive effect of hypoxia. Hypoxia causes increased capillary permeability and pulmonary edema. It has been estimated that 1 minute of oxygen lack may be followed by morphologic changes in the brain and that 3 to 8 minutes of no exchange may be sufficient to produce irreversible changes and death.[7] This damage thus produces the basis for further insult and damage to nervous and cardiac tissues. Hypoxia, thus, has cumulative local as well as central deleterious effects on respiration.

Secondary effect of hypercapnia—increasing toxemia. Blair[3] cited evidence which indicates that hypercapnia is responsible for restlessness followed by mental depression, disorientation, and eventually coma. Hypertension, then later hypotension, vasomotor failure, and circulatory collapse also occur. The early symptoms of these conditions may suggest need for sedation to the unwary. Insult is thus added to injury by treat-

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ing the restlessness of hypercapnia with sedative drugs. Administration of oxygen may reduce hypoxia but will not affect the carbonic acid blood level or elimination of carbon dioxide. Acidosis develops because of hypercapnia, and also because of excess lactic acid produced in the hypoxic tissues.[7] A second vicious cycle thus occurs. As restlessness increases, so does the excess of acid metabolites increase.

SIGNS AND SYMPTOMS OF PROGRESSIVE REDUCTION OF RESPIRATORY FUNCTION

Spencer[12] has emphasized that reduction of respiratory efficiency by these mechanisms is difficult to detect. By contrast, any degree of obstruction of the airway, no matter how insignificant, immediately causes alarm and initiation of conscious efforts which we readily recognize as retractions and use of accessory muscles. Spencer[12] also states that by the time cyanosis usually appears or dyspnea is evident in these conditions, the patient is moribund. He lists the following signs of progressing respiratory impairment.

General signs. The general signs include (1) increasing respiratory rate, (2) decreasing respiratory depth, (3) increasing pulse rate, (4) rising blood pressure (slight to moderate), (5) fatigue—slight pallor, and (6) poor tolerance to examination and nursing procedures.

Specific signs. The specific signs include (1) breathlessness, (2) inability to take a deep breath on command, (3) any change in breathing pattern, and (4) flaring of nostrils.

Visual signs. Visual signs include (1) fluoroscopic evidence of pure pulmonary aeration (diminution or absence of diffuse pulmonary illumination on quiet breathing), (2) usage of accessory muscles (uncommon until very late), and (3) shortened duration of speech.

Audible signs. Audible signs include (1) diminished volume of vocalization—weak, "pitiful" cry, (2) inequality or absence of breath sounds, and (3) decreasing ability to count on one breath.

Measurement sign. A measurement sign to look for is the decreasing vital capacity which is below minimum, normal values.

Spencer[12] goes on to list "signs of progressive asphyxia":

Early signs. The early signs include (1) circumoral pallor—constricted pupils, (2) poor skin color and circulation (mottling of the skin), (3) profuse sweating—especially on the forehead, (4) deterioration of well being and responsiveness (5) progressive restlessness—inability to sleep and apprehension ultimately leading to a panic state, (6) confusion—delirium, (7) marked dilation of nostrils, often with frank dyspnea, and (8) thready pulse—occasionally hypertension followed by hypotension.

Late signs. The late signs include (1) appearance of facial components of breathing, and (2) intermittent cyanosis, hallucinations, panic, coma, persistent cyanosis, and death.

THE CRITICAL IMPORTANCE OF AGE AND SIZE

The factors adversely affecting respiratory physiology may be summarized under the broad head of (1) frank obstruction of upper airway, which produces early dyspnea and late alteration of acid base balance; and (2) progressive loss of respiratory effort, not associated with frank obstruction which does not produce dyspnea until very late (usually irreversible), but which produces chemical changes due to hypoxia, hypercapnia, and respiratory acidosis early and insidiously.

These factors of altered respiratory physiology, whether of the first type characterized by high obstruction, or of the second due to weakness and paralysis or insidious increase of secretions, may operate at any age. Croup and other conditions causing obstructions are more common and acute in younger age groups. Significant mechanical factors responsible for this are well known. The causitive factors of insidious reduction of respiratory effectiveness in the absence of obstruction also operate more frequently and with greater rapidity in small infants, particularly those who are premature or debilitated.

MECHANISM OF ACTION OF TRACHEOSTOMY IN IMPROVING PATIENTS WITH INSIDIOUS RESPIRATORY MUSCLE FAILURE

Tracheostomy improves alveolar ventilation by (1) enhancing removal of secretions, (2) reducing the amount of dead space, and (3) decreasing the resistance to breathing, both on inspiration and expiration (fig. 1).

1. Removal of secretions. Frequent use of suction by nasal catheter in an infant is difficult and inefficient and may actually impair respirations by producing laryngeal edema. Tracheostomy makes possible removal of secretions from the trachea with efficiency and safety. The procedure

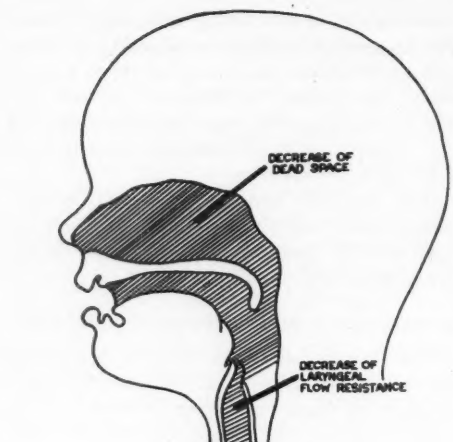

FIG. 1. Mechanism of action of tracheostomy in improving alveolar ventilation.

is thus indicated at times for the relief of progressive asphyxia due to secretions in infants and children, when it is anticipated that intermittent aspiration over a prolonged period will be necessary.

2. Reduction of dead space. The air in the air passages at the end of inspiration does not take part in gaseous exchange and is known as the physiologic dead space. This air must be displaced before respiratory exchange can begin. The value of tracheostomy in reducing the volume of dead space was demonstrated experimentally and clinically by Carter and Guiseffi.[4] Elimination or reduction of dead space increases effective ventilation and may mean the difference between life and death in an infant or child with reduced pulmonary reserve.[10] The effective alveolar tidal volume can be increased by decreasing the dead space fraction of each tidal volume.[1]

3. Decrease in resistance to breathing. Carter and Giuseffi[4] demonstrated by measuring intrapleural pressure that tracheostomy was of value in reducing moderately the resistance to air flow in the trachea by bypassing the glottis. Work necessary to maintain ventilation is reduced. In a critical patient this relief may help to prevent exhaustion. Since this factor of resistance increases geometrically as the area of the passage (glottis) decreases, it is clear that reducing this resistance by bypassing the narrow area as well as reducing friction by shortening the length of the passage may significantly influence survival in the type of patient under discussion.

CASE REPORTS

In each of the 4 cases presented, it was felt that tracheostomy would be beneficial in reducing the respiratory work load by reducing respiratory dead space and resistance to flow, and that tracheostomy would also be beneficial in facilitating removal of secretions. Laboratory guides such as blood pH, CO_2 content, and arterial oxygen saturation were not obtained. These chemical measurements are often of indeterminate therapeutic value and are far less reliable than careful clinical evaluation.[12]

Case 1. J. F. C. was born April 30, 1957, by low forceps delivery following induction of labor because of malignant hypertension in the mother. The birth weight was 3 lb. 14 oz. The infant breathed spontaneously and had a good color and a fair cry immediately after birth. Increasing respiratory difficulty was noted soon thereafter. Severe retraction and wheezing began 12 hours after birth. At 72 hours the infant became somewhat dusky in spite of oxygen and high water concentration in Isolette. A chest film taken on May 3 demonstrated complete atelectasis of the left lung. The clinical impression was that the patient had hyaline membrane disease, and that he was accumulating secretions in the larger respiratory passages. Tracheostomy was performed in the operating room under well controlled conditions. Traction was also applied to the sternum by a towel clip which was hung from the top of the Isolette by an elastic band. Immediate improvement was noted. On May 4 aeration in the left lung was still poor, but on May 6 it was much improved. A chest film on May 14 was essentially negative. The tube was left in place for 10 days, during which time frequent aspiration was necessary. The environment was kept constant with

high humidity. Oxygen concentration was kept below 30 per cent. The patient began gaining weight and made an uneventful recovery. At 1 year of age she appears bright and active.

Although this child did have evidence of some degree of high obstruction, the major indications for tracheostomy were to facilitate removal of secretions from the lower respiratory passages, to guard against aspiration of vomitus and saliva, and to reduce respiratory effort. Because improvement began immediately following tracheostomy, it was thought that this procedure contributed to this infant's survival.

Case 2. Baby boy B was a full term infant born May 27, 1957, by cesarean section because of cord prolapse in the first stage of labor. Heart tones were good throughout delivery. Anesthesia was minimal. At time of delivery the infant was intensely cyanotic and relaxed. He made no effort to breathe, and after a waiting period of 2 to 5 minutes an intratracheal airway was inserted and oxygen was administered under positive pressure. Respiratory stimulants were used and the infant was placed in a Rockette. The color improved but the tube was kept in place for several hours as a precaution. A tracheostomy was done electively because of the baby's inability to use respiratory muscles and because the intratracheal tube had been in the glottis. During the first 24 hours continuous rocking was necessary because of episodes of prolonged apnea, and during the second 24-hour period this artificial respiration was gradually diminished. The Rockette was not completely eliminated until after 72 hours. The tracheostomy tube was removed on the 7th day. There was no further evidence of respiratory insufficiency. The question of neurologic deficit was raised at time of discharge. Development had been retarded to a moderate degree.

Tracheostomy was performed in this infant to obviate high obstruction from edema which might follow insertion of the intratracheal tube and to reduce the respiratory work load. Finally, because of the weakened condition at birth, it was feared that aspiration was a real threat to survival.

Case 3. J. J. was a 4-month-old colored male admitted to the Medical College Hospital on January 23, 1957, following 4 days of severe diarrhea. On admission, he was cyanotic, lethargic, had poor skin turgor, dry mucous membranes, and a sunken fontanelle. Respirations became very poor and periods of apnea ensued. Artificial respiration was necessary. The usual measures for hydration were instituted and because of the critical condition, steroids were given intravenously. Despite these measures, periods of apnea supervened three times during the first morning after admission and it was apparent that the patient's respiratory function was failing. Tracheostomy was, therefore, performed. Immediately thereafter, respirations became much better. There were no further episodes of spontaneous apnea and oral fluids were begun on the 2nd day. Improvement continued and he returned to normal health. The tube was removed on the 5th postoperative day.

Tracheostomy was performed to diminish dead space, thereby increasing the effective tidal volume and to facilitate removal of any aspirated secretion which the child seemed to be unable to swallow. This child was extremely weak and it was felt that reducing respiratory effort by reducing the dead space and respiratory resistance contributed markedly to the patient's survival.

Case 4. D. A. C. was a 3-year-old infant admitted to the Medical College Hospital on September 11, 1957, with a history of progressive paralysis (beginning 5 days before admission) in the legs and becoming generalized. A diagnosis of polyneuritis was made. By September 28 muscle tone had diminished to the point of there being very little movement of any part of the body. She became unable to swallow and episodes of coughing, choking, and cyanosis occurred. A chest film revealed a patchy, ill defined, increased density in the midportion of the left lung field compatible with an aspiration pneumonia. On September 30 tracheostomy was performed following preoperative insertion of intratracheal airway. There was gradual improvement of both pulmonary status and underlying disease. On October 10 respirations were stronger and the lungs appeared essentially clear by x-ray. The tube was removed on October 19. Improvement continued and she was discharged ambulatory on December 4.

Although tracheostomy had nothing to do with improving the underlying neurologic disease it did contribute in a major way to survival in this child as it allowed proper respiratory tract toilet during the period when the child could not do this adequately on her own. Effective tidal volume was increased at a time when respiratory excursions were lessened.

TECHNIQUE

No attempt will be made here to give in detail the technique of doing a tracheostomy because it is adequately described in many surgical texts and journals. Attention is called to certain important features of this procedure that apply to infants and small children. Of greatest importance, the procedure should, whenever possible,

be done under well controlled conditions in the operating room with an anesthetist present. An intratracheal airway should be inserted through which adequate respiration with manual pressure assistance where needed can be maintained throughout the procedure. In the infant and young child the pulmonary apices may be distended and may bulge high into the neck, meeting in the midline. Because of this, exposure of the trachea is made more difficult, and, at the same time, the danger of accidental nicking of the pleura with the scalpel is increased. Holinger and his associates[6] warns against incision of the posterior wall in the small, soft trachea of infants and children. Putney[9] prefers incision of two rings rather than removal of a segment of a ring for the reason that any excision of the tracheal wall in infants contributes to the development of narrowing of the operative site and may lead to stenosis. The tube should not be placed too low as it might impinge upon the carina, producing partial obstruction of a main stem bronchus. Proper size of the tube is important. It should rest comfortably in the trachea and not occupy the entire lumen. A no. 00 or 0 tube with a short barrel has been found to be most satisfactory in infants. Children 2 years old and over will usually accept a no. 2 tube; children 5 years old, a no. 3 tube; and children 8 years old and over, a no. 4 tube.[11] The tube must be either sutured in place or tied around the neck by several knots that cannot be untied. The skin can be closed loosely around the tube or left open. Postero-anterior and lateral chest films should be taken immediately after the operation to determine the position of the tube in relation to the carina.

AFTER CARE

The period after tracheostomy is performed requires expert management of the young patient as the difficulties in maintaining an airway through such a small tube are many and complex. Inadequate supportive care can lead to grave consequences. The inspired air must be saturated with water vapor to keep bronchial secretions liquefied and to prevent drying of the bronchial mucosa. A Croupette or Isolette will serve this purpose well. We have found that, with little children, these tents make it difficult to keep the body temperature from falling. The use of a good humidifier powered by compressed air is preferable. An obstructing mucus plug should be suspected if dyspnea occurs in spite of aspiration. These will sometimes form at the end of the tube causing intermittent occlusion. If this occurs the tube must be replaced by a clean one. Mucus plugs in the trachea below the tube can be removed by bronchoscopy through the tracheal opening. Removal of crust is facilitated by flushing with a drop of saline each time the suction is used, or once every 30 minutes We have not found Alevaire or other detergents of value. Nelson[8] has used trypsin for those cases having severe crusting Aspiration of secretions should be accomplished with catheters which are kept sterile. The same catheter should not be passed into the nasopharynx and then through the tracheostomy tube. Suction should be applied intermittently and only as the tube is being removed. We use a Y-tube, the open end of which is closed by the thumb for suction Prolonged suction can cause respiratory embarrassment in an infant. Turning the head from side to side will facilitate suction of each main stem bronchus. Insertion and withdrawal of the catheter must be gentle in order to keep irritation of the mucosa to a minimum. The tube should be removed as early as is feasible to avoid formation of granulation tissue in the trachea.

COMPLICATIONS

Most complications can be attributed to faulty technique or inadequate supportive care in the postoperative period. Bigler[2] reported 4 deaths in 62 cases which he attributed to faulty postoperative care. Nelson[8] reported 54 complications and 16 deaths in 310 cases. Six of these deaths were attributed to errors in technique and after care. Ill fitting tracheostomy tubes in infants cause discomfort, irritation, ulceration, and crusting which leads to narrowing of the airway and obstruction by plugs of inspissated mucus.[6]

The occurrence of complications following emergency tracheostomy performed on infants and small children is much higher than following those done electively under well controlled contions. Bleeding postoperatively is usually from the thyroid gland or small vessels of the anterior neck. It is usually easily controlled. Pneumothorax and mediastinal emphysema can occur as a result of damage to the pleura or by passage of air into the mediastinum from the tracheostomy

wound through the fascial planes. Strenuous inspiratory efforts with marked increase in intrathoracic pressure can rupture the mediastinal pleura. Wound infection, persistence of fistula, and esophageal injury are uncommon. No complications occurred in these 4 cases.

SUMMARY AND CONCLUSIONS

Four cases are reported which demonstrate the important effect of tracheostomy on respiratory physiology in infants and young children when insidious respiratory muscle failure is progressing. Tracheostomy is advantageous in reducing the volume of respiratory dead space and decreasing the resistance of flow; it also makes secretions in the lower respiratory passages more accessible. Signs and symptoms of progressing respiratory impairment which are associated with respiratory muscle weakness are outlined. Important steps in the technique of tracheostomy in this age group and the necessity of adequate care following this procedure are emphasized. Complications can be reduced to a mimimum if tracheostomy is carried out under well controlled conditions and observance of good tracheostomy care is maintained in the postoperative period.

It must be stressed that tracheostomy is no panacea for saving seriously ill infants and children. Extreme care must be utilized in selection of patients for tracheostomy under conditions herein outlined. The physician performing a tracheostomy on an infant or small child must accept the grave responsibility of the post-tracheostomy care.

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REFERENCES

1. Affeldt, J. E., Collier, C. R., and Hackney, J. D.: Mechanical treatment of pulmonary failure. Postgrad. Med., *19:* 550, 1956.
2. Bigler, J. A., Holinger, P. H., Johnston, K. C., and Schiller, F.: Tracheotomy in infancy. Pediatrics, *13:* 476, 1954.
3. Blair, N. J.: Tracheotomy in secretional hypoxia. Canad. M. A. J., *72:* 766, 1955.
4. Carter, B. N., and Giuseffi, J.: Tracheotomy, a useful procedure in throacic surgery, with particular reference to its employment in crushing injuries of the thorax. J. Thoracic Surg., *21:* 495, 1951.
5. Fitz-Hugh, G. S., and McLean, W. C.: Tracheotomy: indications and comments. J. Michigan M. Soc., *56:* 1400, 1957.
6. Holinger, P. H., Johnston, K. C., and Anison, G. C.: Tracheotomy. S. Clin. North America, *35:* 21, 1955.
7. Nelson, T. G.: Tracheotomy: a clinical and experimental study, part I. Am. Surgeon, *23:* 660, 1957.
8. Nelson, T. G.: Tracheotomy: a clinical and experimental study, part II. Am. Surgeon, *23:* 750, 1957.
9. Putney, F. J.: Complications and postoperative care after tracheotomy. A. M. A. Arch. Otolaryng., *62:* 272, 1955.
10. Ransdell, H. T., Jr.: Tracheostomy in absence of high respiratory obstruction. Am. Surgeon, *22:* 258, 1956.
11. Shepard, R. M., Jr.: Indications, technique and aftercare of tracheotomy. J. Oklahoma M. A., *49:* 6, 1956.
12. Spencer, W. A.: *Treatment of Acute Poliomyelitis*, Charles C Thomas, Springfield, Ill., 1945.

SPONTANEOUS ESOPHAGEAL CUTANEOUS FISTULA*

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Because it is necessary to have an unusual set of circumstances to bring about a spontaneous esophageal fistula that communicates directly with the exterior and not traversing the pleural space, we have decided that this case warrants reporting. We believe that this is an unusual complication of pulmonary surgery and it illustrates certain principles that must be observed in esophageal surgery.

The first case of rupture of the esophagus was described by Boer Haave in 1724; the first antemortem diagnosis was reported by Meyer in 1858; and the first operative repair in this country was performed by Overholt in 1943.[2]

A review of the literature reveals reports of tracheoesophageal fistulas, or cases of esophageal pleural cutaneous fistulas, spontaneous and otherwise; but it is quite rare to find an esophageal cutaneous fistula reported. In fact, we did not encounter such a report. Furthermore, it is difficult to find cases of esophageal fistulas secondary to a chronic sinus tract as a result of pulmonary surgery whether it be for tuberculosis or other diseases.

A spontaneous rupture of the esophagus is defined by Pate and his associates[3] as "perforation of the normal esophagus resulting from mechanical forces entirely within the patient's own body and divorced from external violence." Perforations of the esophagus may be the result of instrumentation, esophagitis, neoplasia, congenital anomaly, infection, stricture, diverticulum, penetrating trauma, and increased intra-abdominal pressure such as coughing, vomiting, defecation, and so on. Our case was secondary to inflammation; that is, a chronic sinus tract.

The classic clinical picture of spontaneous rupture of the esophagus is one that produces severe upper abdominal and thoracic pain with dyspnea, collapse, and profound shock. This usually follows episodes of increased intraabdominal pressure caused by retching, vomiting, or the like. Our case, in contrast, although due to retching and coughing, caused no shock or untoward symptoms because the leakage from the esophagus immediately escaped externally. Our case presented no problem as to diagnosis, but the problem was that of surgical therapy.

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CASE REPORT

U. F., a 27-year-old white woman, was admitted to the Greenwell Springs Tuberculosis Hospital on April 20, 1949. Admission diagnosis was far advanced pulmonary tuberculosis. Admission x-rays presented a giant cavity off the right hilum with caseous pneumonia in the lower two-thirds of the lung. There were scattered infiltrative lesions throughout the left lung and especially concentrated in the second interspace. The patient was extremely ill, very dyspneic, and had fever of 104° to 105° F. After approximately 6 months of bed rest and streptomycin therapy (this was in the early days of streptomycin and she received streptomycin alone), although her sputum remained positive and she continued to run a low grade temperature, she began to gain weight and improved considerably. The associated disease in the left lung had begun to clear and it was felt at this time that her best chance for survival would result from a right pneumonectomy. The patient continued to remain quite dyspneic and, because of this, it was elected to operate, using the anterior approach. On November 11, 1949, a right pneumonectomy was performed. The lung was firmly adhered to the parietal pleura and it was necessary to do a pleuropneumonectomy. In freeing the lung from the diaphragm there was considerable spillage from contents of the giant cavity. The specimen presented a cavity measuring approximately 7.5 cm. in diameter and was located in the center of the right upper lobe; the lower half of the upper lobe showed extensive infiltration. The lower and middle lobes revealed an extensive caseous pneumonic process. There was very little normal lung tissue in the specimen except at the extreme apex of the upper lobe. Furthermore, on microscopic study, all of the mediastinal nodes presented with the specimen showed tuberculosis.

The patient did fairly well for a few days but then developed a wound infection and the anterior thoracotomy incision dehisced some 10 days postoperatively. Approximately 1 week following this there was evidence of empyema, which required a

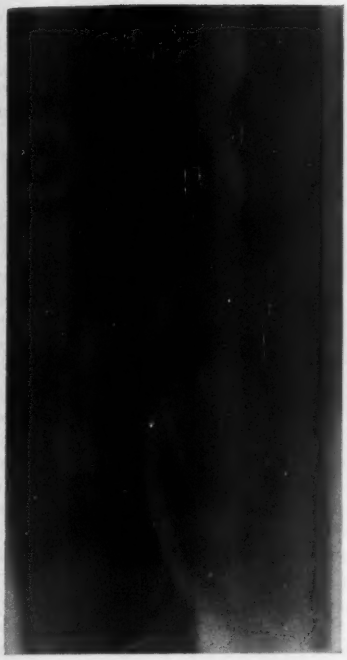

FIG. 1. Preoperative esophagram taken shortly after latest admission which shows a traction type diverticulum.

rib resection with open drainage. Within a few days a bronchopleural fistula was evident as the patient began to expectorate thick purulent and bloody material. It then became necessary to perform almost a total thoracoplasty which was accomplished in three stages. The patient again began to do well except that she always had a small draining sinus which drained anteriorly. The bronchial fistula closed, the patient gained weight, remained afebrile, and continued to improve. It is to be noted that the patient never had a positive sputum or a positive wound culture for tuberculosis following the initial surgery.

She was discharged to her home on April 1, 1950, with a small pleurocutaneous fistula. This was cared for by her private physician. She returned in 1955, at which time a modified Schede procedure was done in an attempt to excise the remaining pleurocutaneous fistula and close a small empyema space. However, there still remained an opening in the anterior chest wall which would, on occasion, drain a few cubic centimeters of purulent material.

In November 1957, while the patient was eating peanuts followed by a drink of water, she choked on the water and noticed that a peanut had come through the sinus opening on her anterior chest wall. Following this, all food that she swallowed appeared on her chest wall.

She immediately returned to the hospital and it was evident that the patient had an esophageal cutaneous fistula. The patient had no fever or untoward symptoms of any kind, but the fistulous opening in the esophagus grew in size and it was obvious on inspection of the anterior chest wall. A gastrostomy was performed with the hope that by feeding the patient *via* this method the abnormal tract would close. Barium swallow at this time led to the assumption that the esophagus had been involved with an inflammatory process and had developed a traction type diverticulum in the middle third and that the diverticulum had ruptured at the time of the paroxysm of coughing and led to the present fistula (fig. 1). After approximately a week it was seen that the fistula still remained open. Under general anesthesia the edges were freed and sutured with 0000 chromic interrupted sutures. However, this did not hold for more than 48 hours and the fistula was again present and continued to grow in size. The wound was packed daily and gastrostomy feedings were continued. The patient gained weight and felt good; however, the fistulous opening in the esophagus was pouting and seemed to be as large as ever.

FIG. 2. Postoperative esophagram which demonstrates a normal caliber to esophagus.

FIG. 3A. Photograph showing a marked deformity of the anterior chest wall; tracheal rings can be seen through overlying skin.

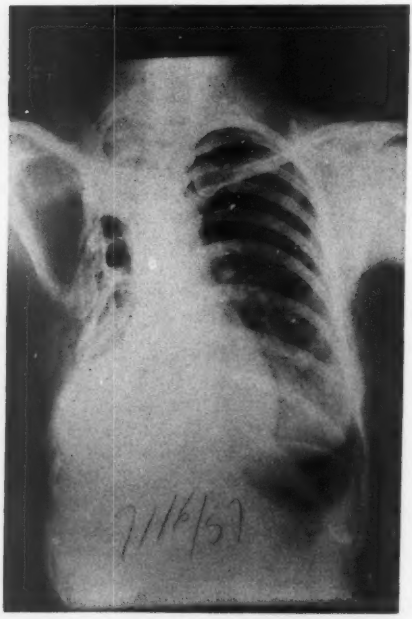

FIG. 3B. Routine x-ray taken prior to development of esophageal fistula shows extent of thoracoplasty which made the anticipated dissection of the mediastinum difficult.

On February 11, 1958, at reoperation under general anesthesia, the esophagus was dissected free approximately 4 cm. above and below the fistulous opening. The entire pathologic area was excised and, with the proximal and distal segments of the esophagus, mobilized. An end to end anastomosis was performed, using one layer of 0000 chromic inverting mucomuscular sutures reinforced with interrupted 0000 silk seromuscular sutures. The wound appeared closed without undue tension. A full thickness pedicle skin graft was then developed from the skin overlying the right breast and was sutured over the esophagus. The remainder of the wound was closed primarily and a small tissue drain was left in its lower end.

The patient made an uneventful recovery and within 1 week was placed on a soft diet. On approximately the 10th postoperative day there was a small granulating area on the medial portion of the wound which showed evidence of some leakage. This was quite distressing. By withholding everything by mouth for 2 days, however, there was never any more evidence of leakage. The patient made a good recovery without further complications and was discharged from the hospital in the 5th postoperative week. At this time it was noted that the patient, for the first time in 9 years, had no draining sinus. She was able to eat a full diet and an esophagram showed a normal caliber to her esophagus (fig. 2).

The delay in attacking the fistula primarily was partly caused by the amount of scar that was seen in this area (fig. 3*A* and 3*B*). While inspecting the patient's anterior chest wall the pulsations of all the pulmonary vessels were readily seen. The location of the bronchus was obvious and it was felt that any dissection in this area would be done with considerable risk of getting into the hilar vessels. As shown previously by many surgeons, however, it is necessary, when one has a fistulous tract such as this, to attack it directly and to do a primary anastomosis of good tissue and not try to close the diseased tissue. It is also to be noted that the pathologic report of the excised esophagus showed only chronic inflammatory tissue with no evidence of neoplasia or tuberculosis.

SUMMARY

1. A case of esophageal cutaneous fistula in the midthoracic region is presented.

2. The lesson learned is that one should make every attempt to close all draining sinuses in the chest as well as elsewhere as it is believed that in nature's attempt to close this sinus, a traction diverticulum developed in the esophageal wall. It was through a rupture of this diverticulum that the fistula occurred.

3. It was only after the esophagus was dissected free to where an adequate layer to layer anastomosis was accomplished with fine sutures that the fistula remained closed.

4. Early attempts at bypassing the fistula by doing a gastrostomy accomplished no change in size in the abnormal opening of the esophagus.

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REFERENCES

1. Kinsella, T. J.: Spontaneous perforation of the esophagus. Am. Surgeon, *17:* 584, 1951.
2. Derbes, V. J., and Mitchell, R. E., Jr.: Rupture of the esophagus. Surgery, *39:* 688, 865, 1956.
3. Pate, J. W., Hughes, F. A., and Patton, T. B.: Spontaneous rupture of the esophagus. Am. Surgeon, *24:* 385, 1958.
4. Graham, E. A.: Editorial comment. In *The 1943 Year Book of General Surgery*, p. 382. Year Book Publishers, Chicago, 1944.
5. Lockwood, T. M.: Acquired traumatic esophago-tracheal fistula. Canad. M. J., *76:* 749, 1957.
6. Abrams, H. S.: Esophagorespiratory fistulae. A. M. A. Arch. Otolaryng., *60:* 371, 1954.
7. Coleman, F. P.: Acquired non-malignant esophagorespiratory fistula. Am. J. Surg., *93:* 321, 1957.
8. DeBakey, M. E., and Heaney, J. P.: Tracheo-esophageal fistula due to non-penetrating injury. Am. Surgeon, *19:* 97, 1953.
9. Meyer, J: Meter Tesseinsung dei Speiseröhe—as quoted by Derbes & Mitchell.

LOCALIZED ARTERITIS AND THROMBOSIS OF THE ILIAC ARTERY ASSOCIATED WITH THE USE OF INGUINAL TRUSS*

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Arterial occlusion due to arterial thrombosis resulting from trauma has been described by Allen,[1] Barker and Hines,[2] Lowenberg,[3] Platt,[4] and Wright[5]. Trauma by crutches, cervical ribs, air hammers, and pressure of malignant lesion has been indicated. Sleeping with the arms hyperabducted has also been listed as a cause of arteritis causing thrombotic occlusion.

Nowadays, the truss has little place in the treatment of inguinal hernia, but an occasional patient still comes to the attention of medical men with the findings of a long worn inguinal support. A search of the literature, however, has shown no case of an arterial lesion attributed to a truss. The following case report presents details of an instance in which truss pressure may well have caused arterial occlusion.

FIG. 1. The iliac and femoral arteries are narrowed. These vessels underlie the usual position of the truss.

CASE REPORT

P. R., a 63-year-old white man, was admitted to Albuquerque Veterans Administration Hospital 10 days after the acute onset of severe burning pain over the dorsum of the right foot and the lateral surface of the calf. The patient had been hospitalized since the onset of pain in another hospital. He was totally unable to walk because of overwhelming pain in the involved calf and foot, and required large doses of narcotics to control the pain. He denied any previous history of claudication, night cramps, coldness, or pain in the right lower extremity. He had had a large right inguinal hernia for 20 years and had worn a truss for 19 years.

Physical examination revealed a large right inguinal hernia. The rest of the positive findings were confined to the right lower extremity. There was no muscle wasting; distribution of hair and skin texture were both normal; the right foot and distal one-third of the calf were cold and blanched; an area of hyperesthesia over the dorsum of the foot and the lateral surface of the calf was present; and no pulse could be palpated in the right lower extremity.

An electrocardiogram was normal and no cause of embolus was found. An aortogram revealed complete arterial occlusion of the right external iliac artery 1 to 2 cm. above Poupart's ligament.

Exploration was performed on April 23, 1958. The initial incision was made on the medial side

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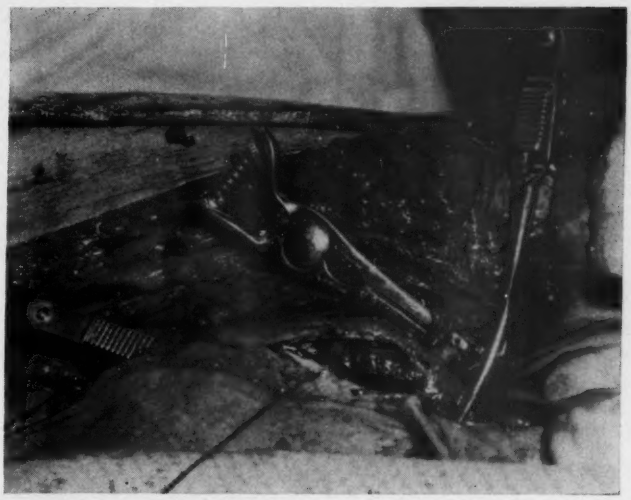

FIG. 2. Thrombus and part of intimal plaque in the opened ileofemoral artery

FIG. 3

of the ankle and showed the posterior tibial artery at the ankle to be a fibrotic band with no lumen.

A second incision was made over Scarpa's triangle. The distal superficial femoral artery was patent and had a normal appearing intima. The femoral incision was continued through Poupart's ligament so that the external iliac artery could be visualized.

That part of the external iliac artery measuring about 3 cm. in length, which had been under chronic trauma from the inguinal truss for 19 years, was flattened (fig. 1). The adventitia was bound down in fibrotic scar tissue. The intima was thickened and a fresh thrombus occluded the lumen (fig. 2). The thrombus was firmly attached to an ulcer on the posterior surface of the artery. The base of the ulcer was atheromatous. Above and below this 3-cm. area the artery appeared normal, with little or no atheromatous change.

Since there was a known occluded posterior tibial artery, it was believed that a major portion of the run-off of the superficial femoral artery was impeded. This opinion was strengthened by the observation of very little back flow coming from the open end of the superficial femoral artery. There was a large, pulsatile back flow, however, from the profunda femoris after removal of the clot which had completely occluded all branches of the common femoral artery.

Endarterectomy of the thrombus and affected intima was performed under general heparinization. The thrombus extended some 5 cm. into the

FIG. 3. Arteriogram shows good flow from the common femoral through the profunda femoris and down to the popliteal. Blood flow from the iliac to the profunda femoris had been found occluded by clot at operation.

external iliac artery, 5 cm. distally into the superficial femoral artery, and about 2½ cm. into the profunda artery. Upon reversing the heparinization with protamine the superficial femoral artery promptly clotted, probably due to lack of run-off. The circulation continued strongly, however, through the iliac, common femoral, and profunda femoris arteries.

The inguinal hernia, which was large and sliding, was repaired.

The patient returned for a femoral arteriogram 2 months postoperatively (fig. 3). The right iliac and profunda femoris arteries are patent with a well established collateral circulation. The patient walks without pain and recently walked four miles on a fishing expedition without inconvenience.

SUMMARY

A 65-year-old man who had worn a truss daily for 19 years developed sudden occlusion of the right iliac artery which extended into both the superficial and deep femoral arteries.

At operation, an old localized atheromatous ulcer on the posterior surface of the external iliac artery, and arteritis with recent thrombosis, were found immediately subjacent to the area of truss trauma. This lesion may have been initiated by the truss. Endarterectomy and thrombectomy were performed. Because of the poor run-off of the superficial femoral artery, it occluded postoperatively. Excellent flow was reestablished through the common femoral and profunda femoris arteries, with nearly normal function of the involved extremity resulting.

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REFERENCES

1. Allen, E. V., Pratt, J. H., and Thompson, G. J.: Segmental compression of iliac artery by a metastatic malignant lesion: report of case. Proc. Staff Meet. Mayo Clin., *22:* 525, 1947.
2. Barker, N. W., and Hines, E. A., Jr.: Arterial occlusion in the hands and fingers associated with repeated occupational trauma. Proc. Staff Meet. Mayo Clin., *19:* 345, 1944.
3. Lowenberg, E. L.: Acute traumatic arterial thrombosis of the extremities. Virginia M. Month., *67:* 630, 1940.
4. Platt, H.: Occlusion of the axillary artery due to pressure by a crutch: report of two cases. Arch. Surg., *20:* 314, 1930.
5. Wright, I. S.: The neurovascular syndrome produced by hyperabduction of the arms; the immediate changes produced in 150 normal controls, and the effects on some persons of prolonged hyperabduction of the arms, as in sleeping, and in certain occupations. Am. Heart J., *29:* 1, 1945.

ADENOMATOUS POLYP OF THE DUODENUM: REPORT OF TWO CASES*

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Benign tumors are rarely encountered in the small intestine and are least common in the duodenum. River and his associates[1] made an extensive review of the literature through December 1954 and discussed at length 1399 benign neoplasms of the small intestine, including 20 new cases. A smaller series of cases from the Mayo Clinic was discussed separately. In their large series, 227 cases were adenomas and 170 were polyps. These authors also state "that it is reasonable to believe that a majority of the tumors described simply as polyps are in reality polypoid adenomas." Seventy-two of the adenomas were located in the ileum, 73 in the duodenum, 47 in the jejunum, and the remainder in multiple locations. Only 18 of the 170 cases discribed simply as polyps were located in the duodenum alone.

SYMPTOMATOLOGY

Many small intestinal tumors are either asymptomatic or cause vague symptoms of pain, nausea, and vomiting which may be related by the physician to other organs or which may cause the diagnosis of dyspepsia or psychoneurosis to be made. Obstruction, often due to intussusception, and bleeding are the most common complications.

Duodenal tumors are usually small and are not as likely to cause obstructive symptoms as lesions lower in the small intestine. If obstructive symptoms do occur they are usually intermittent, probably owing to the liquid nature of the duodenal contents. Symptoms of duodenal tumors are not characteristic and may mimic duodenal ulcer or other diseases of the upper abdomen. Bleeding is probably the most frequent complication. Consideration of the possibility is the most important factor in diagnosis. In River's review[1] of 1014 cases in which the tumor was found at operation, only 79 were correctly diagnosed preoperatively.

Two additional cases of true adenomatous polyps of the duodenum are presented below.

CASE REPORTS

Case 1.† A 28-year-old white woman was admitted on April 16, 1953, with a history of four episodes of abdominal pain in the preceding 6 months. This pain was cramping in nature, radiating from the epigastric area to the right upper quadrant, and almost continuous. It was associated with nausea and vomiting on one occasion. There was no relationship to food or position. Each episode lasted 4 or 5 days. The patient was free of symptoms between episodes. She had noticed no change in stool color. Physical examination showed no evidence of abdominal tenderness, distention, or mass. A gastrointestinal series prior to admission to the hospital showed a filling defect in the first part of the duodenum which was thought to represent a polyp (fig. 1).

On the day following admission, under general anesthesia, the abdomen was opened through an upper right paramedian incision. The tumor was palpable in the first portion of the duodenum. The duodenum was opened on the opposite side from the polyp and it was excised transduodenally. The base was closed with a continuous catgut suture. The longitudinal opening in the duodenum was then closed transversely. This area was covered with omentum and the abdomen was closed in layers with cotton.

The pathology report was as follows: "The specimen consists of a polypoid portion of mucous membrane covered tissue measuring 1 cm. in length and 0.8 cm. in diameter. The sections consist of a polypoid portion of duodenal mucosa. The epithelium is rather thin, the duodenal glands are greatly increased in size and number and constitute most of the polyp. The diagnosis is benign duodenal polyp."

The patient had an uneventful postoperative course and was discharged on the 9th postoperative day. According to her family doctor, she is asymptomatic at the present time.

Case 2. A 30-year-old man was admitted on August 31, 1956, with a history of episodes of upper abdominal pain about once a month for 5 years. This pain was frequently associated with nausea and vomiting. About 12 hours prior to admission he had a rather sudden onset of a more

* From the Department of Surgery, Jefferson Hospital, Roanoke, Virginia.

† This case was operated on by Dr. Hugh H. Trout, Jr., who has given me permission to publish this data for the first time.

FIG. 1. Filling defect in first part of duodenum, thought to represent a polyp.

severe abdominal pain again associated with vomiting. Past history was negative except for history of epileptic type seizures following an episode of meningitis as a child. Physical examination showed generalized tenderness and severe muscle spasm of the upper abdomen. Although there was no free air in the abdominal cavity, the clinical picture was that of a perforated viscus. Surgery was performed shortly after admission.

The abdomen was entered through an upper midline incision. Exploration of the entire viscera was negative except for the sensation of fullness to palpation in the first portion of the duodenum. The pylorus was opened on the anterior surface. A large tumor could then be seen in the first portion of the duodenum. Because of its size and appearance, the tumor was considered malignant and a subtotal gastrectomy was performed, with removal of the duodenum well below the tumor. In the course of the procedure it was noticed that gastritis was present, with oozing of blood from several areas of the gastric mucosa. A Billroth I anastomosis was then performed, and the abdomen was closed in layers with silk.

The pathology report was as follows: "The specimen consists of a lower portion of stomach and 6 cm. of duodenum, together measuring 15 cm. in length. In the duodenum there is a fungating papillary lesion 2.5 cm. in diameter. The center is ulcerated. Sections consist of a portion of duodenum. Arising from the mucosa is a papillary lesion composed of glands similar to normal duodenal epithelium but exhibiting some hyperchromasia. There is no loss of polarity and no evidence of invasiveness or malignant change. The lesion closely resembles a polyp of the colon. The central area of the distal section shows neutrophil infiltration and some ulceration. Sections of the stomach show congestion and round cell infiltration of mucosa and submucosa. The diagnosis is benign polyp of the duodenum and chronic gastritis."

For several days postoperatively, the Levin tube drainage contained more blood than is usual following gastrectomy. This was treated with topical thrombin and Gelfoam powder with a good response. On the ninth postoperative day there was spontaneous drainage of a superficial hematoma and minimal superficial wound separation which did not warrant suturing. On the fourteenth postoperative day the patient began vomiting large amounts of bright red blood. This was treated with thrombin, Gelfoam, and whole blood, again with a good response. Thereafter he improved progressively and was discharged on the 29th postoperative day. He was last seen on June 12, 1957. He complained mainly of an increased number of epileptic seizures which were followed by weakness. The only gastrointestinal symptom was occasional "burning pain" following meals. A gastrointestinal x-ray series showed no additional polyps. There was minimal stenosis at the site of the anastomosis but emptying was adequate.

SUMMARY

Two cases of adenomatous polyps are presented, the first of which was correctly diagnosed preoperatively, and the second of which was found at the time of surgery for what was thought to be a perforated viscus. In the first patient surgery was followed by an uneventful course. The second patient had a very stormy postoperative course, at least in part due to gastritis present on admission. Both of these patients had symptoms compatible with intermittent obstruction and symptoms which could easily be attributed to other organs and diseases. This demonstrates the difficulty in diagnosis of this disease.

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REFERENCE

1. RIVER, L., SILVERSTEIN, J., AND TOPE, J. W.: Benign neoplasms of the small intestine; a critical comprehensive review with reports of 20 new cases. Internat. Abstr. Surg., *102:* 1, 1956; in Surg. Gynec. & Obst., *102:* 1, 1956.

SPONTANEOUS RUPTURE OF RENAL NEOPLASMS: REVIEW AND REPORT OF A CASE OF RUPTURED RENAL CELL CARCINOMA*

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Spontaneous rupture of a renal neoplasm is of sufficient rarity to warrant reporting all cases. Beard,[1] in 1946, reporting on spontaneous rupture of the kidney due to all causes, could find only 42 cases of renal neoplasm rupture in the literature.

Frumkin and Meigher,[2] in 1953, reviewed the previously reported cases of spontaneous rupture of renal neoplasms and reported 2 cases of their own. One was a papillary carcinoma of the renal pelvis and ureter with perirenal hemorrhage, and the other was an angiosarcoma of the kidney with retroperitoneal hemorrhage. They were able to find 6 cases in a review of the literature, these cases being, respectively, a renal fibrosarcoma[6] with spontaneous rupture, 2 renal lipomas[4, 5] with perirenal hemorrhage, 1 intraperitoneal rupture of a Wilm's tumor,[8] and finally, 2 cases of rupture of lipsarcomas of the kidney.[3] Shaw,[7] analyzed 40 reports of spontaneous rupture of the kidney due to all causes. These reports represented "all available papers in the English language." He reported 1 case of spontaneous rupture of a carcinoma of the renal pelvis. All the other cases were of spontaneous rupture due to causes other than tumor.

In all, there have been reported 9 cases of spontaneous rupture of the kidney due to renal neoplasms. No previous case report of rupture secondary to a renal cell carcinoma is known. This case is unusual also in simulating a ruptured aortic aneurysm.

CASE REPORT

R. W., A83103, was admitted April 11, 1958, and died on April 24, 1958. She was a 66-year-old colored woman seen in the emergency room with a blood pressure of 84/60 and a history of sudden onset of constant severe epigastric pain and left upper quadrant pain. She was awakened by the sudden onset of this pain at 1:00 a.m. on April 11, 1958. This was associated with gross hematuria. She complained bitterly of abdominal pain, but was unable to describe its character.

Her past history revealed a subtotal gastric resection in 1954 for a bleeding peptic ulcer. A Billroth II procedure had been performed.

Physical examination revealed a well developed, poorly hydrated colored woman, lethargic, semistuporous, and moaning and groaning with abdominal pain. Her blood pressure was 60/0, with a pulse of 130 per minute and respirations of 40 per minute. Her temperature was 37.5° C. A grade III systolic murmur was present at the third right intercostal space in the midclavicular line. A grade II systolic murmur was present at the apex. Other than diminished expansion, the lungs were clear to percussion and auscultation. The abdomen was distended and tense, with extreme tenderness and marked guarding. No bowel sounds were present. A visible and palpable mass filled the entire left side of the abdomen, left flank, and epigastrium. No ecchymosis was present in the left flank. There was slight pitting edema of both lower extremities. The peripheral pulses were full and equal bilaterally.

She was admitted by the Urology Service with a diagnosis of bleeding from a renal neoplasm. Her blood pressure rose to 130/70 with 500 cc. of blood, and 1½ hours later declined to 60/20. She was lethargic and extremely cold and clammy. Bladder irrigations revealed dark, bloody material with clots. During the next several hours after admission she received 2000 cc. of blood. At cystoscopy, the bladder was normal except for a large amount of blood and blood clots. Retrograde pyelography (fig. 1) revealed a moderately large and deformed left renal pelvis, without extravasation. A continuous stream of blood, 40 to 45 drops per minute, appeared from the left ureter. There was very poor function of the right kidney with indigo-carmine, and no function on the left. Despite 2500 cc. of blood she remained hypotensive, with a blood pressure of 70/0 and volume packed cells of 29 volumes per 100 ml. A consultation was obtained from the General Surgery Service.

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FIG. 1. Retrograde pyelogram showing a large and deformed left renal pelvis without extravasation.

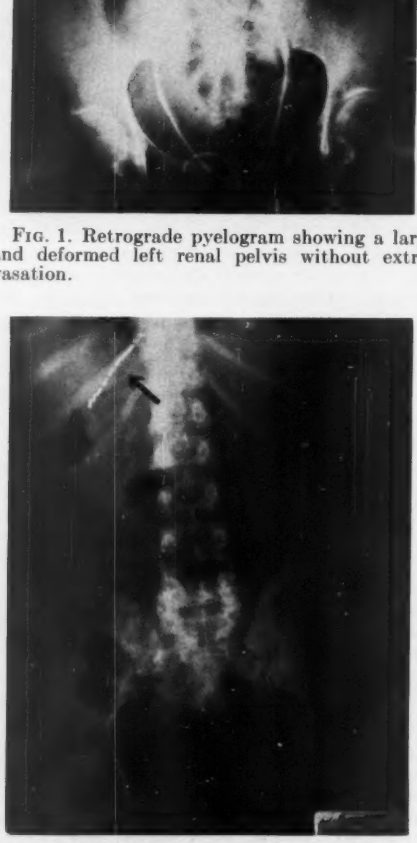

FIG. 2. Scout film of abdomen showing a large mass in the left upper quadrant displacing the silver clips of the previous operation to the right.

An x-ray of the chest showed aneurysmal dilation of the arch of the aorta and moderate cardiomegaly. A scout film of the abdomen revealed a large mass in the left upper quadrant, displacing silver clips from her previous gastric resection to the right. The left psoas shadow was obliterated (fig. 2).

FIG. 3. See text for description

She was accepted on the Surgery Service with a preoperative diagnosis of ruptured abdominal aortic aneurysm. An exploratory laparotomy was performed through a midline incision from xiphoid to pubis. There was approximately 2000 cc. of free blood in the peritoneal cavity, with a 1500-cc. retroperitoneal hematoma presenting in the lesser omental sac. The transverse colon was pushed markedly to the right and inferiorly. The distal abdominal aorta was mobilized and control obtained. The incision was extended into the left thorax through the eighth intercostal space and

the diaphragm divided. The descending thoracic aorta was then mobilized and controlled. The mass was entered and it was apparent that the hematoma was secondary to a ruptured kidney and not a rupture of the abdominal aorta. The left kidney was enlarged 1.5 times, and contained a soft neoplastic mass in almost its entire upper and middle two-thirds. There was complete disruption of the renal capsule in the area of the neoplasm anteriorly. The vascular pedicle was intact. The kidney itself lay in a large mass of retroperitoneal blood clot. Ecchymosis and hemorrhage extended from the diaphragm retroperitoneally into the pelvis and parailiac area. Left nephrectomy and splenectomy were performed with evacuation of the retroperitoneal hematoma.

An intercostal catheter was inserted for postoperative drainage of the left hemithorax. The diaphragmatic, thoracic, and abdominal wounds were closed. Two large Penrose drains were placed in the left subdiaphragmatic space and brought out through the left flank. She received a total of 3500 cc. of blood during the procedure, and at its termination her blood pressure was 120/80. A tracheotomy was performed on the 2nd postoperative day because of excessive tracheobronchial secretions. Her blood pressure postoperatively ranged in the hypertensive levels between 160/110 and 180/110. On the 4th postoperative day she was taking liquids by mouth and had a bowel movement. She was afebrile and taking soft diet on the 5th postoperative day. The blood urea nitrogen was 126 mg. per 100 ml. on the 9th postoperative day and she was lethargic and disoriented. An electrocardiogram revealed evidence of uremic pericarditis or an antero-lateral infarction. Despite all measures she died on April 24, 1958, with progressive lethargy, weakness, and uremia.

Pathology. Pathologic examination of the left kidney (no. 389042) revealed a 15- by 9-cm. kidney which had been previously incised and was grossly distorted by necrosis and apparent tumefactions throughout its cut surface (fig. 3). It appeared that the entire upper pole of the kidney had been replaced by a neoplastic growth, the center of which was hemorrhagic with disruption of the renal capsule. There was gross blood and blood clot in the calyceal system.

Microscopic sections of the kidney showed a malignant neoplastic growth composed of alveolated clusters and cords of cells with comparatively small central nuclei and cytoplasm, some of which were granular and some clear. Large portions of the tumor were necrotic and there was hemorrhage surrounding the kidney tissue itself. Some of the tumor also was hemorrhagic. The histologic appearance was characteristic of renal cell carcinoma (figs. 4 and 5).

Necropsy report. Positive findings on necropsy, April 25, 1958 (no. 58-174) consisted of a healed

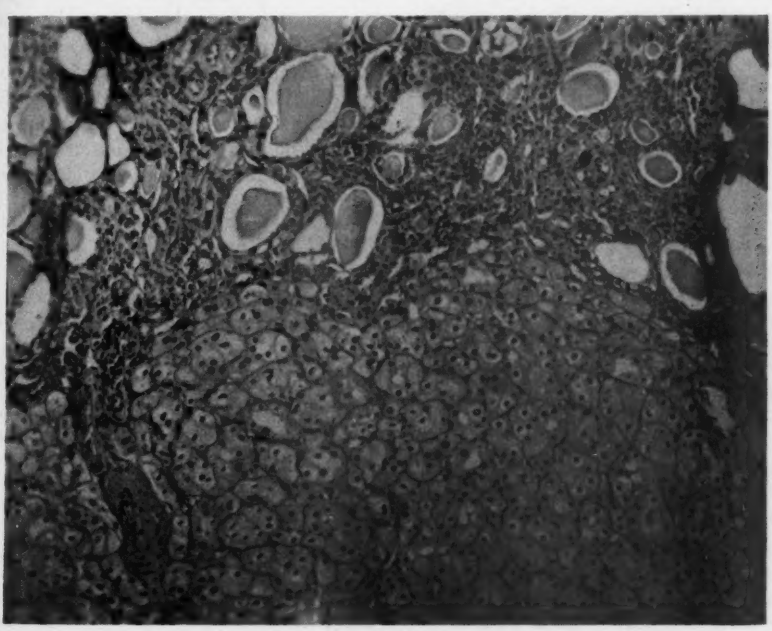

FIG. 4. See text for description (×125)

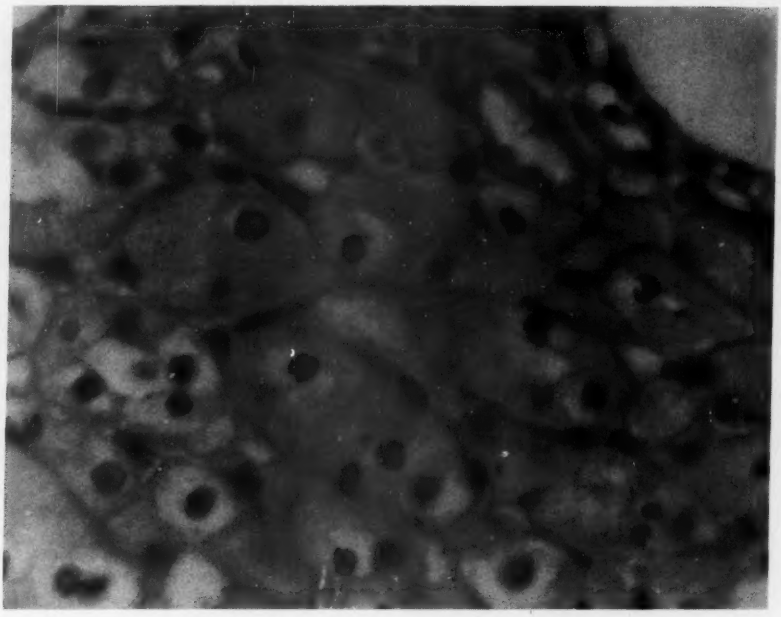

FIG. 5. See text for description (×600)

45-cm. thoracoabdominal incision. There was a considerable amount of organizing blood clot and apparent necrotic tissue in the left retroperitoneal space. The left kidney and spleen were absent. The left pleural space was normal other than some filmy adhesions at the previous operative site. Marked congestion and edema of the bases of both lungs were visible grossly. The remaining right kidney weighed 400 gm. The lower pole was occupied by a 6- by 6-cm. tumor mass. The superior pole of the right kidney contained another tumor mass which measured 2- by 2-cm. Only a small amount of pale renal parenchyma remained which was not involved with this apparent neoplastic process. There was no evidence of distant or local metastases.

Microscopic sections revealed renal cell carcinoma involving the right kidney, pulmonary edema, and congestion.

DISCUSSION

This is believed to be the only reported case of spontaneous rupture of a renal cell carcinoma with hemorrhage. Despite gross hematuria, this patient was operated upon with a preoperative diagnosis of ruptured abdominal aortic aneurysm. Her symptoms were that of an acute abdominal catastrophe and shock, with a progressively enlarging mass in the left flank. A left thoracoabdominal approach gave excellent exposure of the operative area and also enabled proximal and distal control of the thoracic and abdominal aorta. Retrograde pyelography failed to reveal extravasation or sufficient evidence to warrant a diagnosis of ruptured renal neoplasm preoperatively. The presence of bilateral renal cell carcinoma without evidence of any other metastatic focus warrants considerable speculation.

SUMMARY

There have been 9 previously reported cases of spontaneous rupture of renal neoplasms with no known report of a case secondary to a ruptured renal cell carcinoma. A case is reported of spontaneous rupture of a renal cell carcinoma presenting as an acute abdominal catastrophe and simulating a ruptured abdominal aortic aneurysm.

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ADDENDUM

Since preparation of this paper a case of rupture of a hypernephroma, presenting with similar symptoms but secondary to minor trauma, has been reported by I. El-Toraei, Cairo, Egypt (J. Urol., *80:* 297, 1958). The above case remains the only known instance of spontaneous rupture occurring in a renal cell carcinoma.

REFERENCES

1. Beard, D. E.: Spontaneous rupture of the kidney. South M. J., *39:* 780, 1946.
2. Frumkin, J., and Meigher, S.: Spontaneous rupture of kidney tumors. Ann. Surg., *138:* 275, 1953.
3. Grauhan, M., and Hellriegel: Rupture of kidney with lipoangiosarcoma. Ztschr. Urol., *35:* 409, 1941.
4. Herman, L.: Massive spontaneous hemorrhage into and around parenchymal lesions of the kidney. J. Urol., *59:* 544, 1948.
5. Jentzer, A.: Grave hemorrhages provoked by a solitary lipoma having perforated the capsule of the superior pole of the kidney. Urol. & Cutaneous Rev., *52:* 612, 1948.
6. Powell, T. O., and Clark, J. E.: Renal sarcoma: report of a case with spontaneous rupture of the kidney. J. Urol., *62:* 751, 1949.
7. Shaw, R. E.: Spontaneous rupture of the kidney. Brit. J. Surg., *45:* 68, 1957–1958.
8. Tanner, C. H.: Intraperitoneal rupture of a Wilms tumour. Brit. M. J., *2:* 714, 1943.

BOOK REVIEWS

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

Operative Surgery, Vol. 8; *Neurosurgery, Eyes, Ears, Nose, and Throat.* Edited by CHARLES ROB, M.C., M.Chir., F.R.C.S., AND RODNEY SMITH, M.S., F.R.C.S. Butterworth & Co., Ltd., London, 1958.

This volume is characterized by a systematic description of selected operative procedures of the specialties listed in the title. Pre- and postoperative care, operative procedures, and complications are concisely discussed. In detailed drawings, major steps and/or land marks of anatomy are adequately shown to further add to the value of this text.

The selections of operations appear to vary greatly as to purpose within the different subdivisions. The section concerning neurologic surgery pertains to those procedures which would most likely be of interest and of need to a general surgeon in a remote community, whereas many of the operations described in the other sections would only be performed by the specialist in that field. However, this variance of purpose by the contributors and editors does not detract from the value of this volume.

JOHN O. SHARRETT, M.D.

The Cervical Syndrome, Ed. 2. By RUTH JACKSON, B.A., M.D., F.A.C.S. American Lecture Series, Charles C Thomas, Springfield, Ill., 1958.

This monograph, now in its second edition, presents a comprehensive coverage of a complex subject which is seen with increasing frequency. The author has expanded the sections on radiographic evolution of the cervical spine and whiplash injury as a causative factor in the cervical syndrome.

Every physician can prosper by the concise, sound approach Dr. Jackson uses to explain the basic principles causing cervical root symptoms.

JOHN O. SHARRETT, M.D.

Psychological Stress. By IRVING L. JANIS, PH.D. John Wiley & Sons, Inc., New York, 1958.

There have been few significant efforts on the part of investigators to render an understanding of the dynamics of human responses to surgical stress. Much research has dealt with this problem in children, yielding, for the most part, only a descriptive analysis. Adults have been studied either as individuals or as statistical phenomena. Dr. Janis does both in this book and attempts a progressive presentation of the theoretic implications concerning the dynamics of human adjustment to stressful life situations; in this case, surgery.

The book is in two parts. The first deals with the psychoanalytic treatment of a patient preoperatively, during operation, and postoperatively. The author delineates the stresses and the patient's responses in each period and examines them microscopically. His exposition of the psychologic defenses used to meet surgical stress is excellent. These defenses include, chiefly: denial, isolation, regression, repression, displacement, and rationalization. Under surgical stress the patient will have aroused in him many of his infantile fears, particularly those of bodily damage, death, and abandonment. When the patient overreacts (*e.g.*, no fear or excessive fear), repressed infantile fears are involved.

In part 2 the author utilizes intensive interviews of surgical patients and questionnaire surveys of young people postoperatively to study behaviorally (1) the fear of bodily damage and (2) externalized anger. Both situations offer management problems on surgical wards. In general, his hypothesis may be stated as follows: when a patient exhibits a high degree of fear during the "threat" (preoperative) period, he will manifest an intense fear of bodily damage during the "impact" and "postimpact" (operation and postoperative) phases. Should the patient show an extremely low degree of fear during the "threat" phase, then he will demonstrate anger toward the authorities in control in the subsequent phases. Where the patient manifests a moderate degree of fear in the preoperative phase, little emotional disturbance is to be expected during crisis. These generalizations are descriptive, but show predictability, a factor which adds scientific validity.

The author discusses "unrepression" (the patient's reporting of derepressed genetic material) which he calls the "paradoxical effect of stress." This reviewer does not consider it to be a "paradoxical" phenomenon. From the latter's experience in his psychoanalytic work, "unrepression" is observed when patients meet stressful situa-

tions, such as death in the family, separation, frustration, surgery, or a major dental procedure. It is common knowledge that psychoanalytic treatment is predicated on an optimal degree of anxiety within the patient. It is true that extremes of effect or anxiety do make the patient more defensive, but surgery as a rule has not been implicated.

Dr. Janis has an excellent grasp of the literature and his bibliography is quite adequate. His style is somewhat belabored as he develops his hypotheses from his clinical material, but this is acceptable as a means to a teaching end. Although the investigation is approached through surgery, the book would be most useful to those in the fields of psychology and psychiatry, and to the psychiatrically well read surgeon. For the busy surgeon, chapters 6 through 10 are recommended. Clinically and theoretically, these chapters present the patient's reactions which the surgeon observes—and at times understands, at times does not. They serve to clarify for him any perplexity he may have about his patient's emotional responses.

NATHAN SCHNAPER, M.D.